Anæsthesia

JOURNAL OF THE ASSOCIATION OF ANÆSTHETISTS
OF GREAT BRITAIN & IRELAND.

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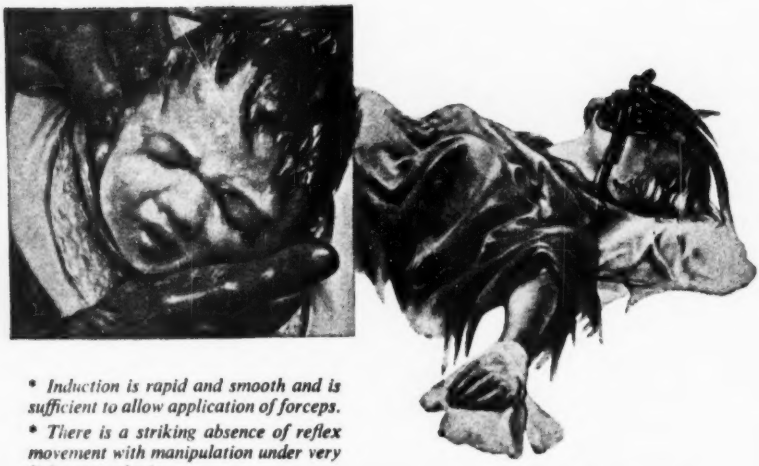
'Fluothane' for Obstetric Anaesthesia



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* *Canad. Anaes. Soc. J. (1960), 7, 109*

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THE ASSOCIATION OF ANÆSTHETISTS
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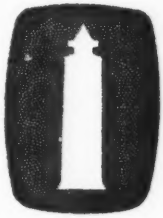

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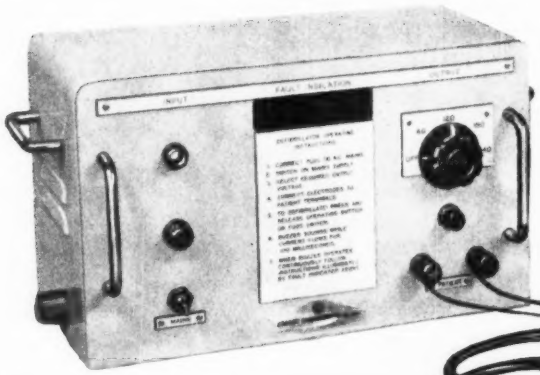

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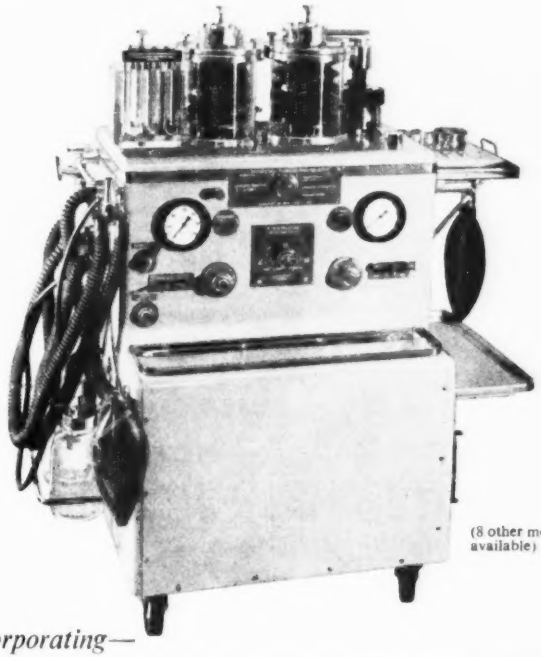

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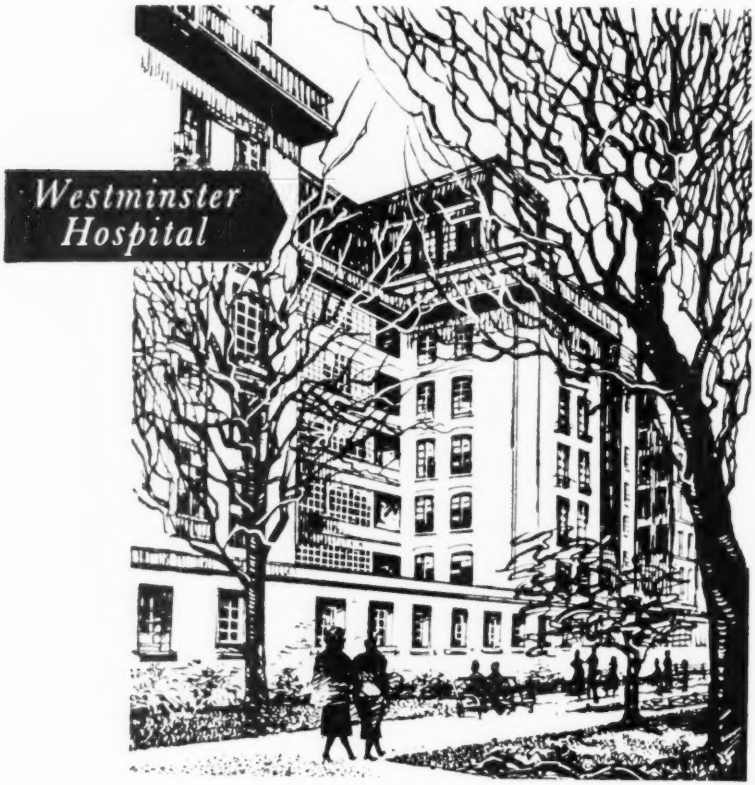

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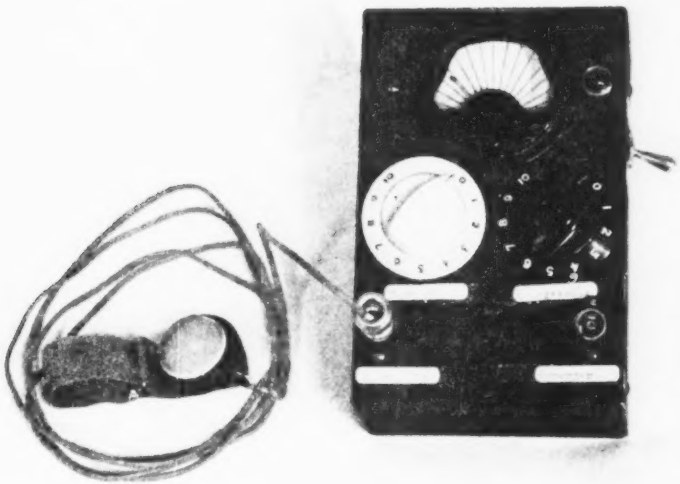

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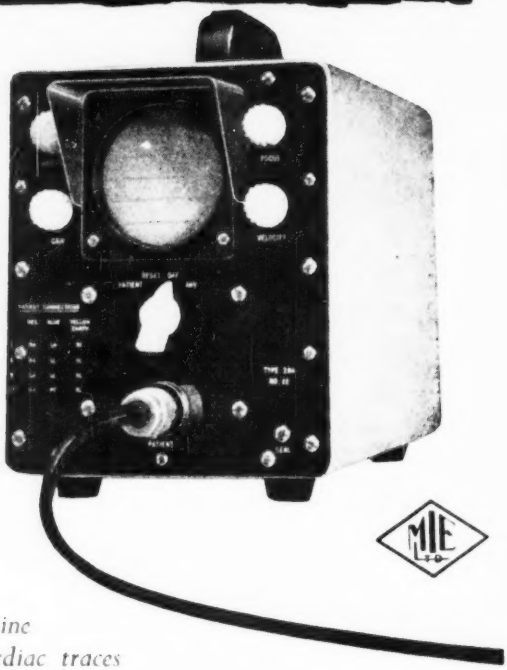

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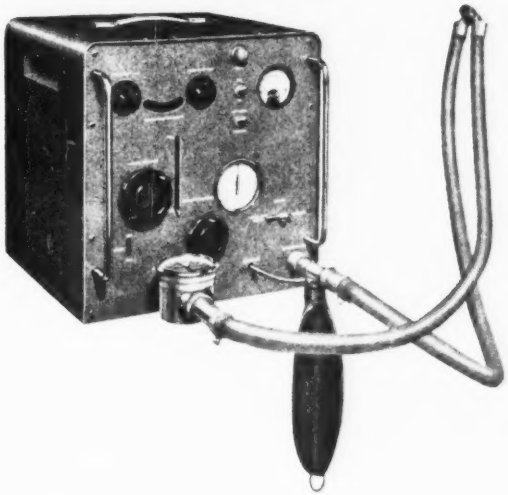

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EDITORIAL

In the July issue of *Anæsthesia*, a short history of the Research Fund of The Association of Anæsthetists of Great Britain and Ireland was given, and the means whereby anæsthetists could obtain research grants were indicated.

The *type* of research as applied to anæsthesia is of the first importance and readers are strongly advised to read the article on this subject by Dr Henry K. Beecher of Boston, USA*. He points out that the specialty needs investigators capable of working in the basic sciences. If these are not forthcoming, anæsthesia will become parasitic depending on scientists in other fields for fundamental developments. New concepts are the essential need for real growth, but all too many 'research workers' are bogged down in perfecting details of apparatus etc, which may be useful temporarily but can never lead to real progress. Anæsthetists may be discouraged from taking up a piece of definite research because they feel that they are inexperienced in some aspect of it, such as statistics. Their work can always be 'vetted' by a statistician or other expert, but 'courses for training in research methods' without a specific object in view, are seldom profitable. Another fallacy is that expensive and complicated equipment is necessary for research. Far from this being the case, the acquisition of such apparatus may become an end in itself and how the said devices are to be used then becomes a real problem. Beecher considers that no anæsthetist should be allowed to obtain complex equipment until he has demonstrated that he can make a significant contribution to knowledge with a notebook, a pencil, an idea and

*J. Amer. med. Ass. (1960), January 30, vol 172, p 449

such simple apparatus as he can build himself. If he shows himself capable of doing this, his clinical work should be adjusted so that he has sufficient freedom to develop his idea.

These thoughts are worthy of close consideration, as unfortunately some of the 'research' now being done is of very doubtful value. On the principle that example is better than precept, we venture to call attention to the first article in our July issue. Here is a piece of research well planned, well carried out and well presented which is of significant value.

A review of the cardiovascular effects of halothane

JOHN W. DUNDEE, MD, PhD, FFARCS

Department of Anæsthetics
The Queen's University of Belfast

GERALD W. BLACK, MD, FFARCS

Royal Belfast Hospital for Sick Children, Belfast

Halothane has been used in clinical anæsthesia for over three years, but there is incomplete agreement on its cardiovascular effects and many conflicting reports have appeared in the literature. It is difficult to evaluate many of the conclusions drawn from published work owing to the number of variables present in the studies, *e.g.* pre-anæsthetic medication, use of other anæsthetic agents, variations in respiratory pattern and the effects of surgical intervention. In this communication, an attempt is made to review the present day knowledge of the cardiovascular effects of halothane and to endeavour to reconcile some of the apparently contradictory findings. It is necessary, first of all, to refer to some of the actions of halothane on other systems, as these modify its effect on the circulation.

It was appreciated early that some degree of respiratory depression was inevitable during halothane anæsthesia, particularly if an opiate was given as pre-anæsthetic medication[1]. This must lead to hypercarbia with a resulting effect on peripheral resistance and circulating catechol amines. Controlled respiration, initiated to compensate the depressant action of the halothane, can affect venous return and arterial blood pressure.

While anæsthesia with cyclopropane or ether is accompanied by a rise in circulating catechol amines, this is not observed with halothane[2]. Hence, the stimulating action of adrenaline and nor-adrenaline on the heart muscle and blood vessels will not be manifest during the use of this drug. The pin point pupils frequently found during anæsthesia and the failure of halothane to produce a rise in blood sugar[3], support the finding[2] that the sympatho-adrenal response is not increased by halothane.

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Alterations in heart rate and rhythm will be discussed first, followed by the action of halothane on blood pressure.

HEART RATE AND RHYTHM

There is no consistent pattern of heart rate changes in man during halothane anæsthesia. Bradycardia is a common, but not invariable, finding, and may be accompanied by a shift of the pacemaker and atrio-ventricular nodal rhythm[4 5 6], indicative of vagal inhibition. These changes may be due to a relative predominance of vagal activity in the presence of a reduced sympathetic response. They are more prone to occur with high inspired concentrations of the drug, but, unlike the ventricular arrhythmias to be discussed later, they are not initiated by hypercarbia and appear to be due to the direct action of halothane on the conducting system of the heart. Should bradycardia be extreme, normal heart rate and rhythm may be restored by the intravenous injection of atropine[4].

When respiration is depressed by halothane, the resultant retention of carbon dioxide may give rise to cardiac irregularities of sympathetic origin[4 5 7]. These include bigeminal rhythm, multifocal ventricular extrasystoles and multifocal ventricular tachycardia. These disturbances of rhythm have not been observed during uncomplicated halothane anæsthesia in the presence of a normal alveolar CO_2 tension[7]. The degree of hypercarbia required to initiate these irregularities, although variable, is high and does not seem to be related to the inspired concentration of the drug[7]. Correction of respiratory acidosis will abolish ventricular arrhythmias even though the inspired concentration of halothane remains unchanged. The P_{CO_2} threshold for the production of arrhythmias is significantly higher with halothane than with cyclopropane[7], and this is probably due to the reduced sympathetic activity with the former drug[2].

The occurrence of ventricular extrasystoles have not often been reported during closed circuit halothane anæsthesia[8 9], which usually causes some degree of carbon dioxide retention. A possible explanation for this anomaly may be that the arrhythmia threshold (P_{CO_2}) is inversely related to the mean arterial blood pressure and so no disturbances in rhythm occur during profound hypotension[7].

Although hypercarbia is probably the most common and frequent instigator of ventricular irregularities during halothane anæsthesia, other causes have been reported. These include endotracheal intubation[10] and surgical stimulation[4 11 12] during light anæsthesia.

Halothane sensitises the heart to the effects of adrenaline[13 14], and ventricular arrhythmias have been noted when a mixture of adrenaline and a local anæsthetic is injected during anæsthesia[15 16]. The infusion

of l-nor adrenaline has a similar effect[17]. It has been noted that adrenaline-induced arrhythmias may be reduced or abolished with ether[15], and animal experiments suggest that perphenazine (fentazin) and other similar anti-adrenaline drugs may prove useful in their prevention[18]. However, one should remember that chlorpromazine may induce severe hypotension during the inhalation of halothane[19].

It is reasonable to assume that all ventricular arrhythmias during halothane anæsthesia, may have a common ætiology, irrespective of their clinical cause. Hypercarbia results in an increase in circulating catechol amines[20]. The breath-holding which accompanies endotracheal intubation during light anæsthesia and surgical stimulation under similar circumstances, may increase sympathetic discharge. These, together with the effects of exogenous adrenaline and l-nor adrenaline, suggest that the sensitisation of the myocardium by halothane to the effects of these amines, is the principal factor in the production of ventricular arrhythmias.

The accompanying table is of value in understanding the fundamental differences in the actions of cyclopropane, halothane and ether on cardiac rhythm[21].

	CYCLOPROPANE	HALOTHANE	DI-ETHYL ETHER
Sensitivity of myocardium to adrenaline	+	+	−
Arrhythmias with normocarbia	+	−	−
Arrhythmias with hypercarbia	+	+	−
Increases in plasma catechol amines			
NORMOCARBIA	+	−	+
HYPERCARBIA	+	+	+

The exact clinical significance of these ventricular arrhythmias is not fully known. Some anæsthetists ignore them and in some centres, halothane anæsthesia is not considered a contraindication to the use of adrenaline[22]. However, a premature ventricular contraction must lead to decreased coronary perfusion and it seems illogical, since they are preventable, that they should be allowed to occur, particularly in poor risk subjects.

Marked bradycardia, with P wave changes, is an expression of relative vagal preponderance and an indication for the use of atropine. On the other hand, ventricular irregularities are manifestations of sympathetic activity and in their presence, the abolition of restraining vagal tone (by atropine) may precipitate ventricular fibrillation[23 24]. It is, therefore, essential that all changes in cardiac rate and rhythm during halothane anæsthesia must not be treated indiscriminately with atropine.

BLOOD PRESSURE CHANGES

Like its effect on heart rate, the action of halothane on the arterial blood pressure is somewhat variable, although some degree of hypotension is a common finding[4 25 30]. The degree of fall in blood pressure is usually related to the depth of anæsthesia[26] and the inspired concentration of halothane. Unfortunately, these generalisations are not universally applicable, and severe falls in pressure can occur during very light anæsthesia[25] and with the head-up posture[15 31]. Reports suggest that severe hypotension is not common during the use of halothane in children[28 32], perhaps due to their faster heart rate.

When the vagotonic effects of halothane are marked and the accompanying bradycardia is severe, some rise in blood pressure can be expected following the injection of atropine, but this does not generally affect the hypotension of deep anæsthesia. A similar improvement sometimes results from the use of gallamine.

Of more importance is the profound cardiovascular collapse and even cardiac arrest, which has been reported when halothane is used with controlled respiration[26 27 33]. A common feature in some of the cases, was the administration of halothane resulting in either marked respiratory depression or inadequate muscular relaxation. One can assume that the concentration of halothane in the anæsthetic circuit is high, but the actual uptake by the patient may not be great due to depressed respiration. When artificial respiration is commenced, with or without a muscle relaxant, the blood level will suddenly rise and these catastrophes could be due to an overdose of halothane. Whether vagal inhibition or a direct depressant effect of halothane on the heart muscle is the cause of the cardiac arrest is debatable. These cases illustrate that the administered concentration of halothane is little guide to the expected effect on the cardiovascular system, without consideration of the respiratory exchange. It is possible that the increased intrathoracic pressure from controlled respiration may play a part in these events and halothane may abolish the compensatory mechanisms which operate in the Valsalva manœuvre.

Halothane will potentiate the hypotensive action of the methonium compounds[34], trimetaphan[35] and chlorpromazine[19]. Severe collapse has been reported following the combination of d-tubocurarachloride and halothane[4], but the frequency or clinical significance of this incompatibility is not known.

Because of its vagolytic action, gallamine seems to be the relaxant of choice for long operations. The use of intermittent doses of suxamethonium, with their resultant muscarinic effect on the heart, is not without danger during halothane anæsthesia and sudden cardiac arrest has resulted from this combination.

All clinicians are agreed that halothane produces a marked peripheral vasodilatation.

It is of interest to consider the theories that have been advanced to explain halothane-induced hypotension. Although the drug suppresses sympathetic activity, the site or sites of such depression are difficult to define in man. Animal experiments have suggested splanchnic vasodilatation and ganglionic block[13], but these findings have not been confirmed by other workers[34] who felt that central vasomotor depression was a more likely explanation for the hypotension. However, since halothane augments the effect of ganglion blocking drugs, it probably produces some degree of ganglionic blockade.

In common with most anæsthetics, halothane exerts a direct depressant effect on the isolated myocardium of the dog[34]. It should be remembered that reductions in the output of the heart-lung preparation are not comparable with those in the intact animal, as the former determinations do not take into account the compensatory reflexes of the circulation produced by the sympatho-adrenal system. This is illustrated by the fact that, although ether depresses the isolated myocardium of the dog, in the intact animal cardiac output is increased, due to the inotropic effects of reflexely released adrenaline and nor-adrenaline[36 37 38]. Owing to suppression of sympathetic activity such compensatory mechanisms are not effective during halothane anæsthesia.

Many studies have been made of the effect of halothane on cardiac output in man, but the results are somewhat conflicting, and difficult to interpret in view of the multiplicity of variable and complicating factors[27 28 39 40]. In general, the findings suggest that clinical concentrations of halothane cause a moderate decrease in cardiac output, but the reductions may be extreme when high concentrations and controlled respiration are used[27]. It has been suggested that the percentage fall in cardiac output in man during halothane anæsthesia, is not greatly in excess of the accompanying reduction of oxygen consumption produced by the drug[39 41 42 43] and thus not indicative of severe myocardial depression.

It would appear that reductions in cardiac output produced by clinical concentrations of halothane, are not excessive and are no greater than those found with other anæsthetics. However, they may be significant in so far as they are not antagonised to any great extent by increases in circulating catechol amines. It is suggested that the essential difference between the effects of halothane and those of other volatile agents on the circulation, may lie in the degree of tone of the peripheral blood vessels.

Preliminary plethysmographic studies reveal marked vasodilatation of the limbs, and reductions in limb vascular resistance of the order of

50 per cent have been observed[44]. Decreases in the total peripheral resistance have also been found to accompany a reduction in blood pressure[27]. A direct local inhibitory action of halothane on smooth muscle may contribute to the above changes[45].

Sensitisation of baroreceptors by halothane must be considered in relation to its hypotensive action, since this is a factor which may play a part in the blood pressure fall and bradycardia associated with chloroform[46]. Although halothane sensitises pulmonary stretch receptors no such effect has been demonstrated on the carotoid sinus[47].

SUMMARY

This paper reviews present knowledge of the cardiovascular effects of halothane.

Arrhythmias and changes in pulse rate can be of two types. Bradycardia and other vagal effects due to a direct action of the drug on the heart predominate during the induction period. Halothane sensitises the heart to the action of adrenaline and the presence of factors which increase sympathetic activity (*e.g.* respiratory depression with hypercarbia and the use of exogenous adrenaline or noradrenaline) will predispose to ventricular arrhythmias.

A variable degree of hypotension is a common finding during halothane anæsthesia. The balance of evidence supports the absence of enhanced sympathoadrenal activity associated with some reduction in cardiac output, and peripheral resistance as the cause of the lowered blood pressure.

References

[1] BRYCE-SMITH, R. and O'BRIEN, M. D. (1957). Some observations on fluothane. *Proc. Roy. Soc. med.*, *50*, 193.

[2] PRICE, H. L., LINDE, H. W., JONES, R. E., BLACK, G. W. and PRICE, M. L. (1959). Sympatho-adrenal responses to general anæsthesia in man and their relation to hæmodynamics. *Anesthesiol.*, *20*, 563.

[3] HUNTER, A. R. (1959). Halothane and the blood sugar. *Brit. J. Anæsth.*, *31*, 490.

[4] JOHNSTONE, M. (1956). The human cardiovascular response to fluothane anæsthesia. *Brit. J. Anæsth.*, *28*, 392.

[5] STEPHEN, C. R., GROSOKREUTZ, D. C., LAWRENCE, J. H. A., FABIAN, L. W., BROUGEOIS-GAVARDIN, M. and COUGHLIN, J. (1957). Evaluation of fluothane for clinical anæsthesia. *Canad. Anæs. Soc. J.*, *4*, 246.

[6] ROBSON, J. G. and SHERIDAN, C. A. (1957). Preliminary investigation with fluothane. *Curr. res. Anesth.*, *36*, 62.

[7] BLACK, G. W., LINDE, H. W., DRIPPS, R. D. and PRICE, H. L. (1956). Circulatory changes accompanying respiratory acidosis during halothane (fluothane) anæsthesia in man. *Brit. J. Anæs.*, *31*, 238.

[8] MARRETT, H. R. (1957). Halothane: Its use in closed circuit. *Brit. Med. J.*, *ii*, 331.

[9] POPE, E. S. (1957). Halothane: A report on its use in twenty-five selected cases of pulmonary or cardiac disease. *Anæsthesia*, *12*, 405.

[10]DELANEY, E. J. (1958). Cardiac irregularities during induction with halothane. *Brit. J. Anæsth., 30*, 188.

[11]AVIPOR, D. (1960). Halothane: Some difficulties and dangers on its administration. *Anæsthesia, 15*, 11.

[12]SHERIDAN, C. A. and ROBSON, J. G. (1959). Fluothane in obstetrical anæsthesia. *Canad. Anæs. Soc. J., 6*, 365.

[13]RAVENTÓS, J. (1956). The action of fluothane—a new volatile anæsthetic. *Brit. J. Pharmacol., 11*, 394.

[14]HALL, K. D. and NORRIS, F. H. (1958). Fluothane sensitization of dog heart to action of epinephrine. *Anesthesiology, 19*, 631.

[15]BRINDLE, G. F., GILBERT, R. G. B. and MILLAR, R. A. (1957). The use of fluothane in anæsthesia for neurosurgery: A preliminary report. *Canad. Anæs. Soc. J., 4*, 265.

[16]MILLAR, R. A., GILBERT, R. G. B. and BRINDLE, G. F. (1959). Ventricular tachycardia during halothane anæsthesia. *Anæsthesia, 13*, 164.

[17]PRICE, H. L. (1959). Quoted by Aviodo, D. M. Cardiovascular effects of some commonly used pressor amines. *Anesthesiology, 20*, 71.

[18]DOBKIN, A. B. and PURKIN, N. (1959). The effect of perphenazine on epinephrine-induced cardiac arrhythmias in dogs: 1 – anæsthesia with fluothane and the fluothane-ether azeotrope. *Canad. Anæs. Soc. J., 6*, 243.

[19]BRENNAN, H. J., HUNTER, A. R. and JOHNSTONE, M. (1957). Halothane: A clinical assessment. *Lancet, 2*, 453.

[20]MILLAR, R. A. (1960). Plasma adrenaline and nor-adrenaline during diffusion respiration. *J. Physiol., 150*, 79.

[21]BLACK, G. W. (1959). Cardiovascular effects of halothane in man: MD thesis, The Queen's University of Belfast.

[22]D'ARCY, E. J., HOLMDAHL, M. H. and PAYNE, J. P. (1959). The pharmacology of halothane in man. *Brit. J. Anæsth., 31*, 424.

[23]JOHNSTONE, M. (1952). Cardiology of anesthesia. *Curr. Res. Anesth., 31*, 325.

[24]POOLER, H. E. (1957). Atropine, neostigmine and sudden deaths. *Anæsthesia, 12*, 198.

[25]PITTINGER, C. B., CULLEN, S. C. and WATLAND, D. C. (1957). Observations on a new anesthetic agent, fluothane. *Arch. Surg., 75*, 339.

[26]HUDON, F., JACKQUES, A., CLAVET, M. and HOUDE, J. (1957). Observations cliniques sur l'anesthesie au fluothane. *Canad. Anæs. Soc. J., 4*, 207.

[27]WYANT, G. M., MERRIMAN, J. E., KILDUFF, C. J. and THOMAS, E. T. (1958). The cardiovascular effects of halothane. *Canad. Anæs. Soc. J., 5*, 384.

[28]MCGREGOR, M., DAVENPORT, D., JEGIER, W., SEKELJ, P., GIBBONS, J. E. and DEMERS, P. P. (1958). The cardiovascular effects of fluothane in normal children. *Brit. J. Anæsth., 30*, 398.

[29]HALL, K. D. and NORRIS, F. H. (1958). Respiratory and cardiovascular effects of fluothane in dogs. *Anesthesiology, 19*, 339.

[30]WYANT, G. M., MERRIMAN, J. E., HARLAND, J. H. and DONALDSON, H. V. (1960). The cardiovascular effects of azeotropic halothane-ether. *Canad. Anæs. Soc. J., 7*, 91.

[31]BURNAP, T. K., GALLA, S. J. and VANDAM, L. D. (1958). Anæsthetic, circulatory and respiratory effects of fluothane. *Anesthesiology, 19*, 307.

[32]LOVE, S. H. S., BLACK, G. W. and CLARKE, R. S. J. (1960). A comparitive study of some of the effects of ether, halothane and halothane-ether azeotropic mixtures on circulation and respiration in children. Paper presented to Second World Congress of Anæsthetists in Toronto.

[33]FOSTER, C. A. (1957). Fatal cardiac arrest with fluothane. *Lancet, 1*, 1144.

[34]BURN, J. H., EPSTEIN, H. G., FEIGAN, G. A. and PATON, W. D. M. (1957). Some pharmacological actions of fluothane. *Brit. Med. J.*, *ii*, 479.
[35]ENDERBY, G. E. H. (1960). Halothane and hypotension. *Anæsthesia*, *15*, 25.
[36]PRIME, F. J. and GRAY, T. C. (1952). Effects of certain anæsthetic and relaxant agents on circulatory dynamics. *Brit. J. Anæsth.*, *24*, 108.
[37]BREWSTER, W. R., ISAACS, J. P. and ANDERSEN, T. W. (1953). Depressant effect of ether upon the myocardium of the dog and its modification by the reflex release of epinephrine. *Amer. J. Physiol.*, *175*, 399.
[38]PRICE, H. L. and HELRICH, M. (1955). The effect of cyclopropane, diethyl ether, nitrous oxide, thiopental and hydrogen ion concentration on the myocardial function of the dog heart-lung preparation. *J. Pharmacol.* and *Exper. Therap.*, *115*, 206.
[39]SEVERINGHAUS, J. W. and CULLEN, S. C. (1958). Depression of myocardium and body oxygen consumption with fluothane in man. *Anesthesiology*, *19*, 165.
[40]PAYNE, J. P., GARDINER, D. and VERNER, I. R. (1959). Cardiac output during halothane anæsthesia. *Brit. J. Anæsth.*, *31*, 87.
[41]KRANTZ, J. C., PARK, C. S., TRUITT, E. B. and LING, A. S. C. (1958). Anesthesia LVII. A further study of the anesthetic properties of 1,1,1-Trifluro-2, 2-Bromochlorethane (fluothane). *Anesthesiology*, *19*, 38.
[42]NUNN, J. F. and MATTHEWS, R. L. (1959). Gaseous exchange during halothane anæsthesia: the steady respiratory state. *Brit. J. Anæsth.*, *31*, 330.
[43]ORTON, R. H. and MORRIS, K. N. (1959). Deliberate circulatory arrest. The use of halothane and heparin for direct-vision intracardiac surgery. *Thorax*, *14*, 39.
[44]MCARDLE, L. and BLACK, G. W. Unpublished data.
[45]BURN, J. H. and EPSTEIN, H. G. (1959). Hypotension due to halothane. *Brit. J. Anæsth.*, *31*, 199.
[46]ROBERTSON, J. D., SWAN, A. A. B. and WHITTERIDGE, D. (1956). Effect of anæsthetics on systemic baroreceptors. *J. Physiol.*, *131*, 463.
[47]WHITTERIDGE, D. (1959). Personal communication.

Halothane anæsthesia for insertion of the Spitz-Holter valve

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The principle of the operation is to drain cerebrospinal fluid from the right lateral ventricle into the internal jugular vein for the treatment of hydrocephalus. A non-return valve is placed subcutaneously behind the right ear, the upper part lying in a groove in the bone. A right-angled catheter leads from the right lateral ventricle to the upper end of the valve while the lower end is connected to a piece of silicone rubber tubing which enters the internal jugular vein, passing down to the superior vena cava or even into the right auricle.

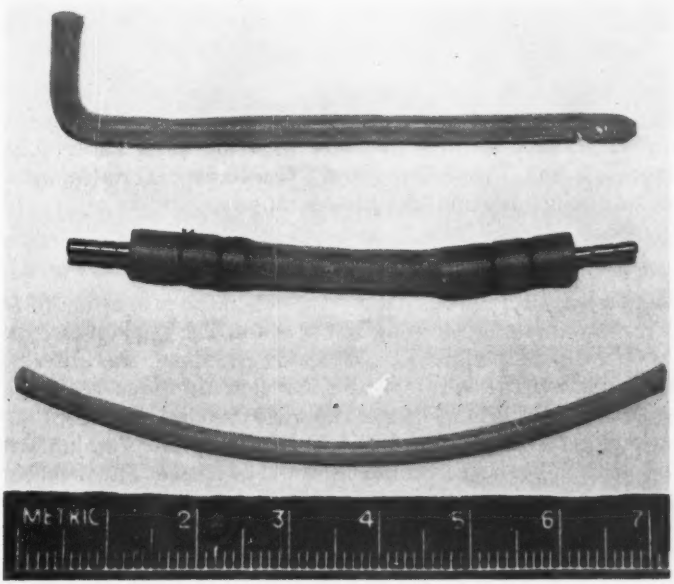

FIG. 1 Spitz-Holter Valve and silicone rubber connections

OPERATION

Operation is undertaken on the supine patient with the head turned about 45° to the left and the shoulders padded in such a way that there are no creases visible in the neck. A curved incision is made behind the ear and the skin and underlying muscle is lifted forwards. The periosteum is incised and a groove is prepared to take the upper metal part of the valve. In the smaller children this groove can be started by bone nibblers at the suture line, but in larger children an electric drill is used. The incision in the neck is made about one or two centimetres above the clavicle and the sternomastoid is split to expose the internal jugular vein. The upper end of the vein is ligated. The right-angled catheter is inserted into the ventricle, the valve fixed in position and the lower length of silicone rubber tubing is passed behind the sternomastoid to appear in the wound in the neck. This tubing, whose length is determined from a straight X-ray film, is then passed down the internal jugular vein to reach its final position either in the superior vena cava or the right auricle. A check film is then taken. If the position of the tube is satisfactory, the wounds are closed in layers.

AGE

In this series of 26 operations, halothane was used as the main anæsthetic agent. The age of the patients was between two weeks and five and a half years. The mean age was nine and a half months.

PREMEDICATION

Atropine was used for premedication on an age basis. Babies 0.2mg, 18 months 0.3mg, 2 years 0.4mg and 5 years 0.6mg. The atropine was given subcutaneously one hour pre-operatively.

ANÆSTHESIA

Induction was with nitrous oxide – oxygen in the proportion of four to three litres per minute, using an infant set. The smallest face mask possible was used to minimise dead space. Halothane was introduced into the mixture after a few breaths and concentrations were rapidly raised to 2.5–3 per cent. A fluotec vaporiser was used in all cases. After a few minutes a Magill armoured tube of appropriate size, lubricated with 4 per cent lignocaine ointment, would be passed. The vocal cords and trachea were sprayed with 4 per cent lignocaine in larger children. No packs were used. Relaxants were not used to facilitate intubation. The Magill tube was then connected to a valveless system consisting of wide-bore corrugated rubber tubing, and secured and held in place with adhesive strapping. Fresh gases are brought in at right angles to

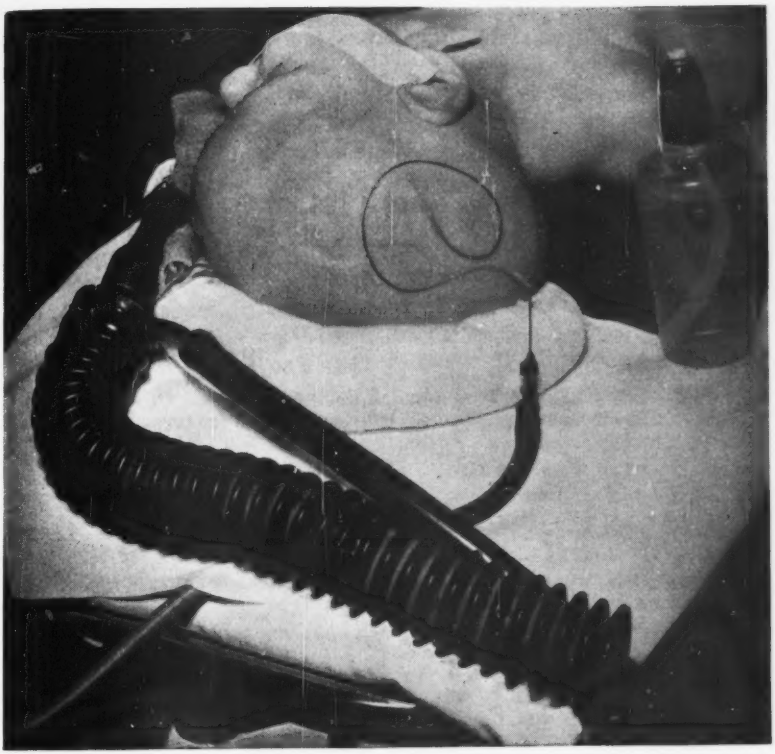

FIG. 2 Valveless circuit

the wide-bore entrance so as not to increase resistance to breathing[1]. High flow rates were used, considerably greater than twice the minute volume, even in the youngest patients[2].

The concentration of halothane had to be at least 1.5 per cent during the preparation of the skin and painting the posterior auricular area with Pig. Mastiche Co. to prevent movement of the child. The concentration of halothane was soon lowered to 1 per cent and later to 0.5 per cent. This latter figure seemed to be enough for the maintenance of anæsthesia. The duration of the operation varied from forty-five minutes to two hours, the average being eighty-five minutes. Scalp vein drips, using the Kempton scalp vein needle, were used in the earlier cases, but in later cases no drip was used. Recovery was rapid except in those cases where much cerebrospinal fluid was lost from the lateral ventricle.

The impression gained in this series was that the intra-cranial tension and the cerebrospinal fluid pressure were lowered[3 4]. Blood loss did not appear to be less than when other anæsthetic agents were used. No marked bradycardia was detected in any of the patients. No patients died during the operative or the immediate post-operative period and no post-operative complications were noted.

SUMMARY

Halothane with oxygen – nitrous oxide has been used in twenty-six cases for the insertion of the Spitz-Holter valve and anæsthesia was uncomplicated in all cases.

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References

[1] CHANDLER, C. C. D. (1958). *Anæsthesia, 13*, 73.
[2] BOOTH, A. J., NISBET, H. J. A. and WILSON, F. (1959). *Anæsthesia, 14*, 355.
[3] BURNS, T. H. S., MUSHIN, W. W., ORGANE, G. S. W. and ROBERTSON, J. D. (1957). *Brit. Med. J., 2*, 483.
[4] HART, W. S. (1958). *Anæsthesia, 13*, 385.

Anæsthesia for surgical emergencies in children

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In recent years, those who work in children's hospitals have witnessed considerable advances in pædiatric anæsthesia. The purpose of this paper is to discuss some of the practical problems which the authors have encountered in pædiatric surgical emergencies whilst they served as Registrars and Senior Registrars from 1955–8. The theoretical aspects of children's anæsthesia and the management of those cases admitted to the Neonatal Surgical Unit are outside the scope of this paper. Furthermore, it has not been possible to include a satisfactory discussion of the complex problems of intravenous therapy in a paper of this nature and any detailed reference to this subject has been omitted.

Reference will be made to 1200 cases which were anæsthetised by the authors after a period of training in the techniques of controlled ventilation, endotracheal intubation, relaxant drugs and nitrous oxide anæsthesia which have been in extensive use by Rees and Stead during the past decade.

THE PROBLEM OF THE FULL STOMACH

The presence of material in the stomach presents the anæsthetist with problems which are somewhat more complex than in the adult.

In early infancy the cough reflex is poorly developed and this will predispose towards the inhalation of food and stomach contents. This may occur prior to anæsthesia and surgery if the infant is severely debilitated and where there is an altered state of consciousness and

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depression of the protective respiratory reflexes. Deaths have occurred in both surgical and medical wards of this hospital under such circumstances.

A male infant, aged four months, was admitted for investigation of Hirschprung's Disease. His abdomen was greatly distended. One week after admission he developed a pyrexia of 103°F and the signs of chickenpox appeared. Three days later he vomited a feed, inhaled the vomited material and died.

It seems likely that the combination of infection and abdominal distension impaired his ability to restore a clear airway.

A male infant, aged seven months, was admitted with a history of vomiting for three days. He was dehydrated and cyanosed and the abdomen was very distended. Whilst preparations were being made for intravenous therapy he vomited a quantity of intestinal contents and died. Post-mortem examination showed the larger air passages to be full of vomitus.

In such cases, it is obvious that even the use of local analgesic techniques will not completely guarantee the safety of the airway and that endotracheal intubation is essential.

It is clear, also, that any additional depression of the state of consciousness and the protective respiratory reflexes in the post-operative period following general anæsthesia must be avoided.

A healthy child aged 5 years underwent adeno-tonsillectomy under deep ether anæsthesia. Post-operatively, bleeding was quite severe and the blood was not replaced. When she returned to theatre she was restless and crying but not fully orientated. The bleeding was arrested under nitrous oxide anæsthesia with relaxant, intubation, and controlled ventilation and at the end of the operation she returned to a similar state of consciousness.

Two hours after her return to the ward she suddenly sat up, vomited, became cyanosed and died. Cardiac massage was too late to be effective. At post-mortem the trachea and bronchi contained large amounts of fluid from the stomach.

In this case, death probably resulted from a combination of factors – aspiration, blood loss, the erect position, the presence of a post-nasal pack, and delayed recovery from the effects of the ether anæsthesia.

Trauma may cause a loss of gastric tone with a delay in emptying time. It may be difficult to determine the time and nature of the last meal and its relation to the time of an accident. A history obtained from the child is often less misleading than one from the parents as many children maintain a closely guarded secrecy about what they eat outside the home.

A child may be unable to tolerate the passage of a stomach tube and such a tube cannot entirely empty the stomach of solid food. The tip of a catheter may come to lie above the level of fluid in the stomach and hence attempts at aspiration should be repeated with the child in a variety of positions.

Such problems may be resolved in various ways, none of which is wholly satisfactory. If the child has eaten within six hours of admission the operation may be delayed if surgical indications permit. It is recognised that such a period of delay does not always ensure an empty stomach and in view of this the surgeon may query the wisdom of waiting at all. However, it is clear that the greater the interval between the ingestion of food and the induction of anæsthesia the less will be the risk.

When there is need for *prompt* surgical intervention, and in all cases where the patient is suffering from an abdominal complaint associated with nausea and vomiting, a stomach tube is passed. This frequently induces vomiting. When this desirable response is not forthcoming, aspiration gives some clue as to the nature of the stomach contents and permits the removal of the fluid portion.

Even these measures do not provide absolute safety. The anæsthetist must always be prepared to cope with vomiting or regurgitation during induction and an effective means of suction should be constantly at hand.

Intubation with a close fitting endotracheal tube provides maximum protection of the airway from inhaled vomitus and if rapidly accomplished greatly reduces the danger. Accordingly, the surgical emergencies in this series have been intubated. The authors have disagreed about the importance of posture during induction. Some of our cases were induced in the steep head-up position, others while lying horizontal.

Vomiting during recovery is no less dangerous than during induction. When the thiopentone, nitrous oxide – oxygen and relaxant sequence has been employed, recovery of consciousness and full reflex activity is almost immediate. Vomiting occurs at the end of the operation, while the endotracheal tube is still in place. Under these conditions the anæsthetist is still in control at this dangerous period and the airway is protected by the presence of the tube. It is our practice to retain the tube in position until it induces a bout of coughing and this may play some part in minimising post-operative chest complications.

Children are not returned to the ward until they have regained a state of full consciousness, that is a state of awareness of environment and of self[1] and are able to converse or respond rationally to questions. We believe that this is especially important in the case of the debilitated child.

PREMEDICATION

There is still some controversy about the ideal method of premedication in children. The child differs from the adult in that he is less able to understand the circumstances which have led to his sudden removal

from the safety of his home. Pain, strange surroundings and various uncomfortable procedures add to his bewilderment.

We believe that sympathy, simple honest explanation and a high standard of nursing care provide a better solution to these psychological problems than heavy sedation. Rees[2] has drawn attention to the child's great fear of abandonment, particularly below the age of five years and to the dangers of the prolonged state of semi-consciousness which may follow the use of large doses of sedative drugs.

Our main efforts, therefore, have been directed towards reassurance of the child. Drug therapy has been limited to the relief of pain and the use of atropine as an anti-sialogogue. Premedication must vary with the weight, age and condition of the patient. If morphine and similar drugs are given in doses calculated on the basis of body weight, the response in the child will in most cases closely resemble that in the adult. Debilitated patients, whether adults or children, require reduced doses.

Morphine is usually avoided under the age of one year, in view of the sensitivity to its action which is exhibited by infants under this age.

Table 1

Drugs used for premedication

Atropine	Neonate	0.16mg (gr1/400)
	1–3 months	0.20mg (gr1/300)
	3–12 months	0.32mg (gr1/200)
	12–24 months	0.40mg (gr1/150)
	Over 2 years	0.60mg (gr1/100)
Morphine	Over 1 year	1.60mg (gr1/40) per stone (0.23mg per kg)
Pethidine	Over 1 year	5mg per stone (0.70mg per kg)

Drugs used during anæsthesia

Thiopentone	Maximum induction dose	20mg per stone (3mg per kg)
Suxamethonium	Initial dose	5–10mg per stone (0.7–1.4mg per kg)
	Incremental dose	1.25–2.5mg per stone (0.17–0.35mg per kg)
D-tubocurarine (over 6 weeks)	Initial dose	2–4mg per stone (0.3–0.6mg per kg)
	Incremental dose	0.25–0.5mg per stone (0.03–0.07mg per kg)
Atropine (to precede Neostigmine)		0.12mg (gr1/500) per stone (0.02mg per kg)
Neostigmine		0.5mg per stone (0.07mg per kg)

In TABLE 1 the doses of premedicant and anæsthetic drugs which we have used in this series are detailed. Whenever possible, our patients have been weighed before operation, as it is considerably more accurate to base the dose upon body weight than upon age in years. In most cases the intramuscular route has been used for premedication. Although this provides a reliable route of absorbtion, it may add considerably to the child's distress if clumsily performed. Experienced

nurses, sharp needles and a small total volume of injection are essential if the child is not to come to theatre – and an intravenous induction – with an exaggerated fear of 'the needle'.

When the patient is shocked or debilitated, however, it is wise to give premedicant drugs by the intravenous route, as absorbtion from the tissues may be delayed. When this well known maxim is ignored, serious consequences may result.

> A boy, aged 13 years, was admitted with a history of colicky abdominal pain for four days. He was seriously ill, dehydrated, with a grossly distended abdomen and absent bowel sounds. Intravenous therapy was commenced with full strength plasma. The premedication was ordered by a junior physician and consisted of atropine gr1/100 (0.6mg) and morphine gr1/6 (10mg) to be given subcutaneously.
>
> This produced no significant analgesic effect prior to operation, when a ruptured appendix with peritonitis were discovered and drained. Fluid therapy was continued during and after operation.
>
> Two hours post-operatively his circulation had improved sufficiently for the morphine to be absorbed from the subcutaneous depot. Drowsiness was followed by severe respiratory depression, the respiratory rate falling to five per minute, with the appearance of cyanosis.
>
> The injection of small doses into the intravenous drip pre-operatively would have allowed the effect of serial doses to be assessed, providing pre-operative analgesia and avoiding the dangerous post-operative respiratory depression.

INDUCTION

Induction by the intravenous route offers undoubted advantages in pædiatric anæsthesia. By comparison, inhalational methods are slow and there may be a prolonged period of semiconsciousness and confusion which may be harmful to the child. Noises grow louder, there is a feeling of nausea, strange gowned figures circulate in strange surroundings and if the mask is applied closely to the face the feeling of terror may be very real indeed.

The adult has welcomed the relief from these discomforts which follows the use of intravenous techniques and there is little logical reason to suppose that the child will do otherwise. Recently it has been suggested that intravenous induction is greatly preferred by children and may play a considerable part in allaying their fears of anæsthesia and surgery[3]. Doughty[4] has also produced evidence that the traditional fear of 'the needle' has been greatly over-emphasised and that it can be overcome by reassurance and careful attention to technique.

We have endeavoured to secure conditions of reasonable quietness in the anæsthetic room and to limit the number of people therein to four – the patient, the nurse who has gained his confidence in the ward, the anæsthetist and an assistant. Masks are not worn. While they wait, the nurse tries to interest the child in a book, which is held in such a position as to obscure the site of injection from the child. After re-

assurance, it is explained that the assistant will hold one of the child's hands and lightly squeeze his arm; that there will be a little scratch and that he will feel sleepy.

A suitable vein is selected at the elbow, the wrist, or the back of the hand. A further warning is given just before the puncture is made – and it is noteworthy that the word 'scratch' conjures up less brutal associations than do 'jab' or 'prick'. The assistant holds the arm firmly to prevent movement and the patient is asked to cough while a small sharp needle is inserted. Under these conditions, a surprisingly high percentage of children will be quite unaware that venepuncture has taken place.

The technique of induction must be modified in certain circumstances, according to the condition of the child, the presence of an intravenous drip and the expected duration of the operation. When the child is reasonably fit, anæsthesia is induced with thiopentone in 2.5 per cent solution, the maximum dose being 20mg per stone body weight (3mg per kg). This is followed at once by either suxamethonium 5–10mg per stone (0.75–1.5mg per kg) or by d-tubocurarine, 2–4mg per stone (0.3–0.6mg per kg). In infants, venepuncture is usually possible using one of the veins in front of the wrist. Even small veins may

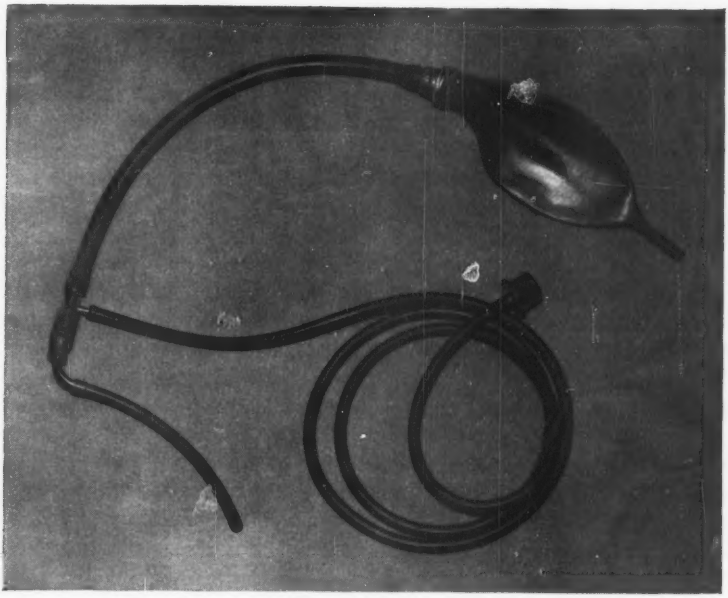

FIG. 1 The Rees modification of Ayre's T-piece circuit

be entered quite easily if a sharp new No 26 needle is employed and a trained assistant holds the wrist in the optimal position, with the skin stretched and the veins congested.

The use of intramuscular suxamethonium prior to endotracheal intubation is considered unwise as the onset of respiratory paralysis may produce marked respiratory insufficiency before the muscles of the jaw and larynx relax sufficiently for intubation to be performed.

Following the injection of the relaxant, the patient's lungs are inflated with a mixture of nitrous oxide – oxygen in equal parts, using a face mask attached to the Rees modification of Ayre's T-piece circuit (FIG. 1). The flow rates used are in excess of twice the child's minute volume. Any difficulty which is experienced in ensuring adequate ventilation is overcome by the insertion of a small airway of the Guedel type. Ventilation is performed as gently as possible to avoid gastric distension.

When relaxation is complete – and this may take two or three minutes in the case of d-tubocurarine – intubation is performed using a rubber Magill endotracheal tube, lubricated with non-analgesic jelly.

TABLE 2 indicates the size of tube required at different ages.

Table 2

AGE	NO. OF MAGILL TUBE REQUIRED
Under 1 year	00 or 0
Between 1 and 2 years	1
2 years	2
Over 2 years	(AGE IN YEARS + 1) ÷ 2

Over 8 years a cuffed tube is used.

A tube which is one size smaller than that anticipated is always to hand

The length of the tube should be such that its distal end comes to lie a short distance below the vocal cords. A rough estimate of the length of an oral tube may be made by measuring one and a half times the distance between the corner of the mouth and the lobe of the ear.

Under the age of ten to twelve months, we prefer to introduce the tube by means of a straight bladed Shadwell Hospital type laryngoscope, which ensures a satisfactory exposure of the cords, especially if an assistant applies gentle backward pressure on the glottis. In older patients the curved Macintosh instrument is used.

The mouth is lightly packed with sterile oiled gauze, which serves to absorb secretions, to block the escape of gases from the larynx and to stabilise the endotracheal tube. The tube is attached to the T-piece circuit and secured with adhesive strapping.

Following intubation, hyperventilation is possible if muscular relaxation is maintained and by this means the tension of nitrous oxide in the blood may be raised rapidly to levels which provide adequate anæsthesia.

MAINTENANCE

Throughout the operation, anæsthesia is maintained with nitrous oxide, five parts to two parts of oxygen. Relaxation is achieved by the use of serial doses of suxamethonium or d-tubocurarine. Measurement of these doses can only be accurate if dilute solutions are used.

RECOVERY

When the operation is completed, spontaneous respiration is allowed to return. If d-tubocurarine has been used, atropine is given intravenously in a dose of 0.12mg per stone (0.02mg per kg). After a period of several minutes, this is followed by neostigmine, 0.5mg per stone (0.07mg per kg). The nitrous oxide is discontinued and the flow of oxygen increased. Suction is applied to the intra-gastric catheter. When spontaneous ventilation is adequate and the patient is coughing on the tube, the pharynx is sucked out, the patient turned on his side with the head down and the tube is removed. Recovery of full consciousness and reflex activity is almost immediate.

When thiopentone has been used in minimal sleep doses and nitrous oxide has been the main agent, there is often a remarkable freedom from pain and discomfort during the post-operative period. This impression is in accordance with the findings of Clutton-Brock[5], who demonstrated the anti-analgesic effect of relatively large doses of intravenous thiopentone in adult patients.

If the child does complain of pain on recovery, it may be relieved by the intravenous injection of pethidine, 3mg per stone (0.5mg per kg) before the patient leaves the theatre. In our experience this has seldom been necessary.

MODIFICATIONS

As has been indicated, this technique may be modified in certain circumstances. There is a large group of patients in whom the clinical condition is difficult to assess from both the surgical and the anæsthetic point of view. In these cases, thiopentone is given slowly and cautiously until sleep is produced, after which intubation and maintenance proceed in the routine manner.

Patients in whom the clinical signs indicate generalised peritonitis, oligæmic shock, or severe deficiencies of water and electrolytes, require careful assessment before operation. When possible, operation is delayed until such deficits have been corrected. In this group there are children who are so gravely ill that, even in the small doses described, thiopentone may cause cardio-vascular collapse. These patients may be induced with nitrous oxide – oxygen mixtures following pre-oxygenation for a period of five minutes. The maximum proportion of nitrous oxide in these mixtures is 80 per cent. Once anæsthesia is

In these cases also we have found that the clinical appearances and the history may be misleading, particularly as regards the probable duration of the state of intussusception. It is difficult to be certain that resection of a portion of gut will be unnecessary. In several cases the condition has been found to be irreducible, even though the history was of but a few hours duration and the clinical and biochemical findings were reasonably satisfactory.

Thus in all cases of intussusception we have considered it advisable to have a reliable intravenous drip set up prior to induction and a supply of blood or plasma readily available. Even if resection is unnecessary, efforts to effect reduction may produce a significant amount of shock, making it difficult to insert a stab drip for resuscitation.

A male infant, aged fourteen weeks, was admitted with a short history of vomiting after feeds. On examination, he was found to be suffering from intussusception. His general condition was good and it was considered that the obstruction was probably of short duration and would consequently prove easy to reduce.

Nevertheless, it was decided to have a cut-down intravenous drip in position before the start of the operation. Laparotomy showed an intussusception extending from the ileo-cæcal region to the hepatic flexure. Attempts to reduce this failed, but as a result of the manipulations the infant collapsed. One hundred ml of plasma were given and the circulation restored, whereupon resection and anastomosis were performed.

If clinical impressions had been allowed to over-rule the practice of having a reliable drip in position before operation, this infant may well have died due to difficulty in inserting a stab drip.

Other surgical conditions in which this technique has been used with success include subphrenic abscess, Meckel's Diverticulum and volvulus. In all these conditions the intra-abdominal pressure may be raised. We believe that the use of relaxants and controlled ventilation greatly facilitates the maintenance of adequate gaseous exchange, especially in infants and young children, where the respiration is predominantly diaphragmatic in type.

Any interference with the movement of the diaphragm will thus cause more embarrassment to the respiration of the infant than of the adult. The adverse effect of abdominal distension is enhanced by the relatively large liver and because the pelvis is unable to accommodate fully its share of the abdominal viscera until after the age of six years.

ORTHOPÆDIC PROCEDURES

Certain orthopædic procedures require a considerable degree of muscular relaxation, for example the manipulation of supra-condylar fractures and the suture of tendons. In such circumstances, the sequence we have described offers advantages to the surgeon and the patient. Of fifty cases of supra-condylar fracture anæsthetised in this manner, only two have required remanipulation.

induced, a relaxant is injected into the drip, the patient is intubated and anæsthesia is maintained in the way we have described. Alternatively, in the event of venepuncture being impossible, it may be wise to use the technique of non-hypoxic induction with nitrous oxide – oxygen and ether[6]. As experience is gained, the need to use this method becomes less frequent.

These techniques have provided satisfactory conditions in a series of over 1200 pædiatric surgical emergencies.

DISCUSSION AND RESULTS

Abdominal emergencies

Seven hundred and eighty cases in this series were operated upon for acute appendicitis, which in this region is by far the commonest emergency operation in children. In general, there is little risk attached to the procedure and this may tend to lull the medical and nursing staff into a false sense of security. The general condition of these patients has frequently proved to be less satisfactory than was indicated by the pre-operative clinical examination. If there has been some delay before operation, the signs of peritonitis may have developed since the child was examined by the surgeon. Hence the wise anæsthetist will try to visit these cases just before they are brought to theatre. On several occasions we have discovered that a child who had been left for some hours was severely shocked and in need of intravenous therapy. We have already described what may happen should the anæsthetist fail to relate the premedication to the condition of the child.

When peritonitis is discovered during the operation, it has been our practice to set up an intravenous drip before recovery from the anæsthetic and to treat the child as a potential paralytic obstruction.

It is clear that patients with appendicitis will vary markedly in their clinical condition and this is borne out by a similar variation in their response to thiopentone. For this reason we consider it wise to limit the dose to that which will produce sleep, or, where the condition is grave, to use one of the modifications we have described. All possible precautions against the aspiration of vomit must be employed. In this series, the operating time has seldom been longer than thirty minutes and we have found suxamethonium to be the most useful relaxant in the majority of cases.

In 131 cases, intestinal obstruction was the reason for operation. The obstruction was due to intussusception in 90 cases, to adhesions in 21, and to irreducible hernia in 20. The mortality associated with intussusception has fallen in recent years, due to earlier diagnosis, careful intravenous replacement of fluid and electrolytes and advances in anæsthesia. Using the techniques we have described, 285 cases have been operated upon at Alder Hey Hospital without fatality[7].

THORACIC OPERATIONS

In this series emergency thoracotomy has been performed either because of traumatic damage to the lung and chest wall or for the drainage of empyema. In both these conditions the use of positive pressure involves the risk of tension pneumothorax. This is especially true where the patient is an infant suffering from staphylococcal bronchopneumonia.

> A male infant, aged five weeks, was admitted suffering from staphylococcal bronchopneumonia. Thirty-six hours after admission he collapsed before being fed. The skin was pale, mucous membranes blue-grey, and there was mediastinal shift to the left with no air entry to the left lung. There were prolonged periods of apnœa.
>
> Intubation was performed with the object of re-expanding the left lung. Positive pressure resulted in a marked mediastinal shift to the right and the presence of a bronchopleural fistula was suspected. This was confirmed by radiographs which demonstrated a left pyo-pneumothorax. The child was transferred to theatre, where drainage of the pleural cavity was performed.

The management of such cases has been discussed in detail elsewhere[8] and attention directed towards the advisability of avoiding positive pressure in this condition until the surgical preparations for thoracotomy are complete. The method of induction has been modified according to the condition of the child and anæsthesia has been delayed until all facilities to cope with this complication were immediately available.

In spite of this danger, positive pressure respiration has been beneficial in certain cases of bronchopneumonia, especially those in which secretions caused a severe degree of obstruction in the trachea and larger bronchi. Such obstruction will result in an increase in the negative intra-thoracic pressure, so that fluids tend to pass from the vessels into the extravascular spaces. In these circumstances, the state of respiratory insufficiency becomes more marked. Hypoxia and carbon dioxide retention develop and contribute towards the state of circulatory inadequacy. By aspiration of the obstructing secretions and the use of intermittent positive pressure respiration, we have been able to reverse this sequence of events in a number of cases.

Traumatic damage to the lungs and chest wall, if severe, will result in both respiratory and circulatory insufficiency. The treatment of this state presents peculiar problems. Frequently the patient has sustained multiple injuries and the clinical picture may be complicated by head injury, severe loss of blood and various visceral and somatic lesions.

Griffiths[9], after considerable experience with such cases, postulates that either as a result of severe paradox or because of respiratory obstruction, there will be an increase in the negative intra-thoracic pressure. Fluid will pass from the vessels into the interstitial spaces, and this will be hastened by the transfusion of excessive amounts of blood.

If these processes are allowed to continue, the lungs will become progressively less compliant. Trauma to the substance of the lung and intra-pulmonary bleeding will also contribute to this end.

Thus it is necessary to correct the factors which cause an increase in the negative intrathoracic pressure. In this connection, intermittent positive pressure respiration is valuable if used at an early stage, before the compliance of the lungs is markedly reduced. Also, it is vitally important to avoid overtransfusion during the treatment of blood loss.

Griffiths suggests that, where such severe injuries exist, intubation and broncial toilet should be performed at an early stage and when a clear airway has been obtained, respiration should be controlled. These measures will counter the mechanisms which result in the accumulation of fluid in extravascular pulmonary tissues. With adequate ventilation, the effects of hypoxia and carbon dioxide retention on the circulation are unlikely to be superimposed upon those resulting from blood loss. Once again, the danger of tension pneumothorax exists when positive pressure is applied to the damaged lung, and facilities for pleural drainage should be immediately available.

In order to ensure the cautious replacement of blood loss, Griffiths advises that at the time of intubation a drip should be erected and while maintaining adequate ventilation, blood should be given rapidly until the blood pressure reaches a relatively safe survival level – say 90mm/Hg systolic pressure. Once this pressure is reached, the rate of transfusion should be slow. A careful watch on pulse and blood pressure will now reveal either that the circulatory improvement has been maintained and that there is no longer need for rapid transfusion, or that there is a sharp fall in the blood pressure, indicating that internal hæmorrhage continues and surgical intervention is urgently required.

NEUROSURGICAL PROCEDURES

While the use of relaxants and controlled ventilation is generally accepted in thoracic and abdominal operations, there is still some controversy over their use in neurosurgery.

This series has included eighteen cases of fractures of the skull and intracranial bleeding, all of which have been anæsthetised by the basic technique we have described or one of the modifications. This method has provided smooth induction, absence of straining and the ability to maintain a light plane of anæsthesia with complete control of the airway. Recovery is rapid and changes in the state of consciousness are not obscured by the use of long acting depressant agents. In older children a respirator with a negative phase has provided satisfactory surgical conditions.

Delayed recognition of the significance of hypertension as a sign of intracranial bleeding led to our only death on the table.

A boy, aged 6 years, was admitted with severe injuries following a road traffic accident. There was a fracture of the right femur, the right popliteal artery was severed and the skin was denuded from both legs. In spite of these injuries, his blood pressure on admission was 120/50, with a pulse rate of 140 per minute. There was no loss of consciousness or amnesia and no neurological signs, although there was a bruise on his forehead.

After he had received two pints of plasma and blood intravenously his clinical condition seemed much better. He remained fully conscious, rational and co-operative. The pulse rate fell to 120 per minute, but the blood pressure rose to 165/95.

During the first hour of operation, these levels were maintained. Their significance was not realised until late in the procedure, when we noticed that the right pupil was dilating. Soon after this, the left pupil enlarged. The pulse rate rapidly became uncountable, both pupils dilated fully and death ensued before surgical intervention was practicable.

OPERATIONS UPON THE EAR, NOSE AND THROAT

Bleeding from the tonsillar bed or from other regions of the mouth and pharynx raises serious problems for the anæsthetist. Danger arises from three main sources – there is the bleeding point in the pharynx; invariably blood has been swallowed and the patient may be suffering from shock.

As most of the blood is swallowed and little appears externally on the sheets or pillows, the amount lost is frequently under-estimated. Occasionally an apparently insignificant ooze continues for a long time and the lack of vigorous bleeding obscures the extent of blood loss.

Furthermore, it is not unknown for a brisk hæmorrhage to occur during the initial toilet and location of the bleeding point. For these reasons, careful pre-operative assessment of blood loss is vital and frequently it is prudent to administer blood before operation – or at least to have everything to hand so that transfusion may begin immediately after induction.

The stomach will certainly contain a large amount of swallowed blood, which the child must not be allowed to inhale. A point less often appreciated, however, is that blood may have already found its way into the lungs, even though the laryngeal and cough reflexes have appeared to be active[10]. This risk is enormously increased if these reflexes have been obtunded by sedation, concurrent injury, or slow recovery from a previous anæsthetic. Hence it is essential to examine the child's chest for evidence of areas of collapse and where indicated to employ bronchoscopy or other means of bronchial toilet before the start of the surgical procedure. Again, it is wise to remember that respiratory insufficiency may play a part in producing the signs of circulatory failure.

Premedication in these cases has been limited to atropine alone,

given usually by the intravenous route. All possible measures have been taken to prevent the inhalation of blood. We believe that the production of apnœa has been valuable in this connection. There is no danger of laryngeal spasm such as may result from the use of inhalational agents alone and which may produce a critical state of hypoxia in the shocked patient.

The apnœic technique has also been used with success in twenty cases of emergency mastoidectomy. In children, infection in this site is particularly likely to be accompanied by marked pyrexia. It is necessary to ensure that the temperature is maintained within certain limits if the dangers of hyper-pyrexia and convulsions are to be avoided. Thus in the ward the nursing staff ensure that the pyrexial child is not encumbered with thick clothing, which in addition to increasing the pyrexia, may restrict breathing.

During anæsthesia, there are many factors which combine to increase pyrexia and metabolic rate and tend to produce the state of demand hypoxia which may well be the cause of convulsions under anæsthesia. Among these factors are the hot humid atmosphere, heavy drapes, atropine premedication, respiratory stimulation with such agents as di-ethyl ether and increased respiratory resistance where spontaneous respiration through a narrow-bore endotracheal tube is permitted. It was under such conditions that convulsions occurred in one of the earliest of our mastoidectomy cases.

> A male infant, aged six months, was admitted with signs of mastoiditis. The temperature on admission was 102°F. Premedication for the operation of cortical mastoidectomy consisted of atropine gr1/200 (0.3mg). After induction with thiopentone and suxamethonium, intubation was performed and an attempt was made to maintain anæsthesia with nitrous oxide – oxygen and ether after the return of spontaneous respiration.
>
> Shortly after the commencement of the operation there was marked twitching of the limbs. The ether tap was turned off and anæsthesia continued with nitrous oxide – oxygen, suxamethonium and controlled ventilation. The rectal temperature was found to be 105°F. As the operation proceeded, this gradually fell to 102.4°F and twelve hours post-operatively it was 98°F, the further course of anæsthesia and recovery being uneventful.

MAXILLO-FACIAL INJURIES

In the majority of these cases, we have been reasonably confident that intubation could be accomplished easily and we have not departed from the basic technique described, except in detail. Occasionally, where the full extent of the injury has not been clear, or where the nature of the deformity has suggested that intubation would be difficult, apnœa has been avoided until an endotracheal tube was in place. In some maxillo-facial injuries, it is wise to perform tracheotomy under local analgesia before induction, but fortunately we have not encountered injuries of such severity.

TRACHEOTOMY

The older and more co-operative child may tolerate this operation under local infiltration analgesia. However, the circumstances in which this operation is usually performed are seldom conducive to co-operation and most children require general anæsthesia.

It is always desirable to secure the airway by endotracheal intubation prior to opening the trachea, even though the larynx will only permit the passage of a relatively small tube. Where hypoxia is severe and the child is unconscious, intubation is possible without anæsthesia. If the child is conscious, it is our practice to deliver oxygen through a face mask for a period of five minutes and thereafter to give a small dose of thiopentone intravenously, followed by suxamethonium. The lungs are inflated with oxygen and the child is intubated.

Even if the airway is only partially restored by a relatively small tube, the advantages to the patient and the surgeon are inestimable. Hypoxia is corrected and the operation can proceed according to a plan. The bleeding from the wound which so tends to obscure the field in the absence of a clear airway is greatly reduced, and it is possible to secure hæmostasis before the trachea is opened.

Once the opening is made, the endotracheal tube is partially withdrawn and the tracheotomy tube inserted. If difficulty is encountered, the endotracheal tube may be restored to its original position until the tracheotomy tube can be satisfactorily introduced. In view of the leak of gases which occurs when the trachea is incised, it is wise to ensure that spontaneous respiration is adequate before this stage in the operation is allowed to proceed. Where it has seemed likely that intubation would be difficult, we have used a non-apnœic technique throughout the entire operation.

CONCLUSIONS

The fate of the patient in emergency anæsthesia depends not only upon the inherent merits of the anæsthetic technique, but also upon certain more intangible factors, such as the supervision, training and experience of the anæsthetist; the co-operation of the surgical and nursing staff; the availability of suitable equipment and the presence of skilled technicians. In all of these respects we have been most fortunate.

The method which we have described has provided the surgeons with satisfactory operating conditions. Intubation, the absence of straining and rapid awakening have contributed to the patients' safety and well-being.

In the past, intubation has been considered a traumatic procedure in young children. With the advent of the relaxants this is no longer true and Rees[11] suggests that this belief should be consigned to the realms of medical mythology. Suxamethonium in particular provides

such ideal conditions that there is no reason why any trauma should be caused to the mouth or larynx. Thirty-six thousand patients have been intubated for surgical purposes at Alder Hey and the Royal Liverpool Children's Hospitals during the past five years and in none of these cases has any serious complication occurred.

Trauma may also be avoided if intubation is performed under deep ether anæsthesia, but we would suggest that movement of the endotracheal tube in the larynx will still occur if spontaneous respiration persists and that these movements may be an important cause of laryngeal irritation. Where the respiratory movements are abolished, the incidence of laryngeal œdema appears to be remarkably low.

SUMMARY

The authors discuss some of the difficulties which may be encountered while providing anæsthesia for sugical emergencies in infants and children. They describe their experiences with the thiopentone, relaxant, nitrous oxide – oxygen and controlled ventilation sequence in this field during the years 1955–8.

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References

[1] COBB, S. (1948). Foundations of Neuropsychiatry, 4th ed., Williams and Wilkins, Baltimore.

[2] REES, G. J. (1958). Modern Trends in Anæsthesia, edited by F. T. Evans and T. C. Gray, 1st ed., 211, Butterworth and Co. (Publishers) Ltd., London.

[3] HODGES, R. J. H. (1959). Annual Meeting of the Assocn. of Anæsthetists of Gt. Britain and Ireland.

[4] DOUGHTY, A. (1957). Anæsthesia for Adenotonsillectomy, *Brit. J. Anæsth.*, *29*, 407.

[5] CLUTTON-BROCK, J. (1959). Annual Meeting of Assocn. of Anæsthetists of Gt. Britain and Ireland.

[6] NISBET, H. I. A. and WILSON, F. (1959). The Induction of Anæsthesia: A Safe Technique for the Novice, *Brit. J. Anæsth.*, *31*, 164.

[7] RICKHAM, P. P. (1959). Personal communication.

[8] NISBET, H. I. A. and WILSON, F. (1958). The Treatment of Acute Respiratory Infection in Infants, *Brit. J. Anæsth.*, *30*, 419.

[9] GRIFFITHS, H. W. C. (1959). Personal communication.

[10] STEELE, C. H. and ANDERSON, J. R. (1950). Tracheo-bronchial aspiration following tonsillectomy with general anæsthesia. *Arch. Otolaryng., Chicago*, *51*, 699.

[11] REES, G. J. (1958). Modern Trends in Anæsthesia, edited by F. T. Evans and T. C. Gray, 1st ed., 208, Butterworth and Co. (Publishers) Ltd., London.

Femoral arterial transfusion

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Arterial transfusion is essentially an emergency procedure. It is obviously unphysiological to force unoxygenated blood into and against the arterial stream and the only indication for its use is a severe hæmorrhagic hypotension that cannot be adequately dealt with by intravenous transfusion. The failure of intravenous therapy may be due to small or constricted peripheral veins, or to the condition known as irreversible shock where compensatory vasoconstriction has failed and in which rapid intravenous transfusion, though possible, has little or no effect on the blood pressure. Whether the arterial transfusion works by increasing the coronary[1], hepatic[2], or cerebral blood flow, there is no doubt of its efficiency in this type of case[3 4].

Cutting down on an artery has two main disadvantages; it takes time and the artery will have to be ligated or sutured at the end of the transfusion. It is better to insert a needle into a large artery and this is no longer considered to be either a difficult or a dangerous procedure by the radiologists, who inject more irritating solutions than blood or plasma into the femoral and carotid arteries and the aorta.

The choice of artery must depend to some extent on the site of the hæmorrhage and the position of the patient, but in general the larger the artery the smaller the risk of spasm or thrombosis. Haxton[3] described a method of percutaneous aortic transfusion that necessitated the patient being in either the lateral or the semi-lateral position. Seeley[5] and Brown[4] recommended the femoral route for really acute emergencies and in neurosurgery, with the exception of those operations performed with the patient in the sitting position, this artery is the most accessible one for the anæsthetist. One case of post-transfusion femoral thrombosis has been reported, but no details were given of the technique which had been used[6].

In a sudden and severe emergency the simplest and the quickest form of treatment is often the best and a femoral artery transfusion can be

set up as quickly and as easily as an intravenous one and without any special equipment. Infiltration with procaine is unnecessary in the anæsthetised or unconscious patient; it wastes time and may make the artery more difficult to palpate. In fact it has not been used in more than 10 per cent of the last 300-odd cases that one of us has anæsthetised for percutaneous carotid angiography and no ill effects have followed its omission[7]. In two of the cases to be described, both thin patients, a standard blood transfusion-giving needle was used. In two of the other cases a 9cm, 18 gauge needle, normally used for drawing up thiopentone, was utilised. The remaining case was transfused through a lumbar puncture needle. The artery was palpated below the inguinal ligament and the needle with a syringe attached was inserted into it. The syringe was then removed and a standard blood transfusion-giving set connected. A Higginson's syringe was attached to provide pressure and speed up the flow. A safety bottle[8] or a roller pump[9 10] and a back-bevelled needle would be safer if immediately available.

Arterial transfusion is usually supplementary to intravenous transfusion and it will rarely be necessary to give more than 1500ml before withdrawing the needle. The effect of arterial transfusion depends more on the route than the quantity of fluid given[3 4].

CASE NO 1

A male, aged 22 years, with an abdominal gun shot wound. On admission he was semi-conscious, the blood pressure was unrecordable, and the signs of intra-peritoneal hæmorrhage were present. Peripheral vasoconstriction was so severe that intravenous transfusion was impossible; even under pressure, blood could not be forced into the long saphenous vein.

Though the radial pulse was impalpable, the femoral could just be felt and a needle was inserted into this artery. 1200ml of blood were injected in ten minutes. This raised the systolic pressure to 80mm Hg and laparotomy was commenced under cyclopropane anæsthesia. The abdomen contained 4½ litres of blood. Unfortunately the hæmorrhage proved uncontrollable and the patient died during the operation.

The autopsy showed that the bullet had divided the hepatic artery as well as lacerating the left kidney.

CASE NO 2

A male, aged 37 years, with a penetrating wound of the thorax. On admission he was cyanosed, dyspnœic and severely shocked, the radial pulse being just palpable but the blood pressure unrecordable. After the aspiration of a right hæmothorax and an intravenous transfusion of 1600ml of plasma, the systolic pressure rose to 70mm Hg.

Right thoracotomy revealed a massive hæmothorax and lacerations of the right upper lobe of the lung and the right atrium. 2000ml of blood were given into the external jugular veins without any improvement of the patient's condition. Intra-arterial transfusion into a brachial artery proved

impossible because of spasm. A needle was then inserted into the right femoral artery and 1000ml of blood given in twenty-five minutes, raising the systolic pressure to 110mm Hg.

In spite of further transfusions, the use of vaso-pressors and hydrocortisone, this improvement was not maintained and death occurred eleven hours after operation. The autopsy confirmed the operative findings and a further hæmothorax was present.

CASE NO 3

A male, aged 18 years, with a severe head injury. A left subdural hæmatoma had been evacuated and a tracheostomy been performed. A caval infusion had been set up because the peripheral veins were so poor.

During the second week his condition gradually deteriorated with frequent right-sided epileptiform convulsions and ventriculography on the fourteenth day showed a ventricular shift towards the right side. A trephine hole was made on the left side and a small extradural hæmatoma evacuated. During the evacuation of a frontal intra-cerebral hæmatoma, there was a very severe hæmorrhage from the sagittal sinus. The systolic blood pressure fell rapidly to below 50mm Hg and even with pressure it was impossible to transfuse the patient rapidly enough through the caval drip. As there were no superficial veins available a needle was inserted into the right femoral artery. It was ten minutes before the hæmorrhage could be controlled and the blood pressure began to rise again. By the end of the operation an hour later, 1200ml of blood and 800ml of plasma had been given intra-arterially and intravenously and the systolic blood pressure had been stabilised at its pre-operative level of 90mm Hg.

The patient's condition continued to deteriorate and death occurred on the seventeenth day. The autopsy showed extensive laceration and necrosis of the left frontal lobe and a large area of necrosis involving the left motor area. An ulcerative tracheitis and a consolidation of the left lower lobe of the lung were also present.

CASE NO 4

A man, aged 21 years, with a compound fracture of the frontal bone, through which brain tissue had been extruded and fractures of the mandible, maxilla, and right tibia and fibula. On admission he was in a state of decerebrate rigidity, hyperventilating and bleeding profusely into the pharynx. Blood and vomit had been inhaled. His systolic blood pressure was 90mm Hg and his pulse rate 140 per minute.

Unfortunately the intravenous drip became displaced during transit. It was replaced in the anæsthetic room, but only 100ml of blood and 450ml of triple-strength plasma had been given when a severe sinus hæmorrhage occurred during the early stages of the operation. The systolic blood pressure fell precipitously to 40mm Hg, the pulse rate slowed to 96 per minute and became irregular and the respiration became gasping. The intravenous transfusion was obviously inadequate and a second transfusion was set up immediately in the left femoral artery. In five minutes the systolic pressure had risen to 65mm Hg and the pulse rate to 160 per minute. 650ml of blood were given intra-arterially and about the same amount intravenously during the next thirty minutes and the systolic pressure was stabilised at 95mm Hg.

Recovery was complicated by a severe uræmia with polyuria and an osteomyelitis of the tibia and it was six weeks before he showed any sign of regaining consciousness. It is still too early to estimate the final degree of mental recovery in this case.

CASE NO 5

A male, aged 18 years, with a severe head injury, fractures of the right clavicle and scapula, a compression fracture of the ninth thoracic vertebra and injuries to the right brachial plexus and brachial artery. A tracheostomy had been performed in a neighbouring hospital where he had been transfused with 1620ml of blood and 800ml of plasma.

On admission he was pale, cyanosed and shocked, with a systolic blood pressure of 85mm Hg and a pulse rate of 160 per minute, and in decerebrate rigidity. There was a huge hæmatoma in the right axilla and the right radial pulse was impalpable.

He was transferred to the theatre for burr holes. At the start of the operation the systolic pressure was 95mm Hg and the pulse rate still 160 per minute. After the incision the systolic pressure fell suddenly to 50mm Hg. As the intravenous transfusion was not running well, a needle was inserted into the right femoral artery. 500ml of blood intra-arterially restored the systolic pressure to 90mm Hg in ten minutes. A total of 1350ml of blood intra-arterially and 500ml intravenously was given in forty minutes, when, at the end of the operation, the systolic pressure was 120mm Hg and the pulse rate also 120 per minute. This patient has made a good mental and physical recovery.

SUMMARY

In very acute emergencies justifying the use of arterial transfusion any long needle can be easily and quickly inserted into the femoral artery and special transfusion apparatus, though an advantage, is not a necessity.

Acknowledgements

We wish to thank Mr G. F. Rowbotham, Mr I. N. Maciver and Mr L. P. Lassman, Consultant Neurosurgeons and Dr B. E. Tomlinson, Consultant Pathologist, for their co-operation.

References

[1] VEAL, R., RUSSELL, A. S. and ASHBURN, F. S. (1952). *Ann. Surg.*, *136*, 485.
[2] DANZIGER, A. (1955). *Lancet*, *2*, 701.
[3] HAXTON, H. A. (1953). *Lancet*, *1*, 622.
[4] BROWN, A. S. (1953). *Lancet*, *2*, 745.
[5] SEELEY, S. F. (1951). *Curr. Res. Anesth.*, *30*, 195.
[6] LIARAS, XICLUNA and LECA. (1949). *Afr. Franc. Chir. P.*, 237.
[7] LASSMAN, L. P. (1959). Personal communication.
[8] MACINTOSH, R. R. and PASK, E. A. (1941). *Lancet*, *2*, 10.
[9] MARTIN, P. (1954). *Lancet*, *2*, 1002.
[10] MELROSE, D. G. and SHACKMAN, R. (1951). *Lancet*, *1*, 1144.

Physiological dead space

Changes during general anæsthesia

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Recent work has demonstrated changes which suggest abnormal distribution of gas and blood in the lungs, as a result of artificial ventilation in patients under general anæsthesia[1]. There was a significant increase in the physiological dead space but little increase in the alveolar–arterial P_{O_2} gradient, implying overventilation of parts of the lung which have a small pulmonary blood flow. However, considerable individual differences were seen.

Howell and Peckett[2] found that the lung compliance decreased during general anæsthesia and muscular paralysis and suggested that this was due to abnormal distribution of gas during artificial ventilation.

This paper reports an investigation into the extent to which these variations in ventilation-perfusion balance alter during the course of a surgical procedure.

Christian Bohr[3] was the first to point out that expired gas consisted of a mixture of two components, one from the dead space, and the other from the alveoli. Thus if we consider the case of carbon dioxide*:

$$V_E . F_{E_{CO_2}} = V_D \times F_{I_{CO_2}} + (V_E - V_D) . F_{A_{CO_2}}$$

Hence:

$$V_D = V_E . \frac{F_{A_{CO_2}} - F_{E_{CO_2}}}{F_{A_{CO_2}}}$$

Measurement of the dead space using the alveolar gas concentration cannot have real meaning in cases of severe pulmonary disease, in which valid alveolar gas estimations by all ventilatory means fail. In other words where there is lack of uniformity of the alveolar air the dead space values will vary greatly. In order to overcome this difficulty Enghoff[5], Riley[6] and other workers, have taken the arterial carbon

*The nomenclature suggested by Pappenheimer[4] will be used throughout this paper

dioxide tension as representative of mean alveolar carbon dioxide tension. Thus the above equation becomes:

$$V_{D\,(physiol)} = V_E \cdot \frac{Pa_{CO_2} - PE_{CO_2}}{Pa_{CO_2}}$$

This expression is thought to give a measurement of that part of the inspired volume which does not reach the diffusing surface of the lung and is known as the physiological dead space.

The physiological dead space can be considered to be made up of two components:

(1) The anatomical (or series) dead space, which is the geometric volume of the conducting airways, and approximates closely to the volume as measured by the single breath analysis of Fowler[7].
(2) The alveolar (or parallel) dead space, which is essentially that gas which ventilates alveoli in excess of perfusion.

The writer has studied changes in the physiological dead space during controlled artificial ventilation in the anæsthetised subject in fourteen patients. The pre-operative dead space of five of these subjects was also measured. In another group of six patients the anatomical dead space was measured using the method of Fowler[7]. The alveolar nitrogen non-uniformity was also studied in this latter group.

METHODS: PHYSIOLOGICAL DEAD SPACE

Nature of operation

Ten of the subjects underwent some form of genito-urinary operation. Of the remaining four, one had a cholecystectomy, one a gastro-

Table 1

Age of patient, nature of operation, position and time interval between samples

CASE NO	AGE	OPERATION	POSITION	TIME INTERVAL (MINUTES)
1	54	Prostatectomy	10° head down	60
2	54	Prostatectomy	10° head down	25
3	79	TUR	Lithotomy	30
4	60	Laparotomy	Supine	30
5	58	Gastro-jejunostomy	Supine	45
6	61	Endarterectomy	R lateral	90
7	68	Prostatectomy	10° head down	60
8	63	Prostatectomy	10° head down	60
9	69	Prostatectomy	10° head down	60
10	48	Cholecystectomy	Supine	30
11	69	Total cystectomy	10° head down	120
12	62	Prostatectomy	10° head down	60
13	63	Prostatectomy	10° head down	60
14	69	Nephrectomy	L lateral	45

jejunostomy, one an endarterectomy and the fourth a laparotomy (TABLE 1).

Pre-operative measurement of physiological dead space

The estimation was carried out with the patient lying at rest in bed, unpremedicated, supported by one pillow. The nature of the operation having been explained previously, a nose clip was applied and the patient requested to breathe room air in and out through a Ruben non-return valve. The latter was firmly supported in the mouth by the standard rubber mouth piece, care being taken to ensure that there were no leaks, the expirate was passed through a calibrated dry gas meter and out into the atmosphere through a two-way tap. The rate of respiration and minute volume were recorded and when these had become steady the patients expirate was switched to a Douglas bag by turning the tap. The expirate was collected over a five minute period. During the middle two minutes 10ml of blood were withdrawn from the brachial artery. The oro-pharyngeal temperature of the subject was noted at the time of sampling.

Anæsthetic technique

Though the premedication was not standardised the majority of the patients received pethidine 100mg and atropine sulphate 0.6mg intramuscularly one hour pre-operatively. Induction was performed with a sleep dose of thiopentone followed by suxamethonium 50mg intravenously and a cuffed endotracheal tube was passed orally after topical analgesia with 4 per cent lignocaine spray. No 10 tubes were used in males and No 9 tubes in females. Anæsthesia to the point of apnœa was maintained with d-tubocurarine and intermittent thiopentone with nitrous oxide 75 per cent – oxygen 25 per cent. The patient was completely apnœic throughout the operative procedure and was ventilated by means of a pump, the rate and tidal volume of which could be preset and maintained at a constant level. The pump used was a modification of the Coxeter-Mushin bag, the excursion of which could be altered by varying the pivot of a lever. A Ruben valve was used as an inspiratory and expiratory valve for the bag.

Mixing of inspired and expired gases was prevented by means of a solenoid operated valve designed by Hill and Hook[8] and actuated from a cam. The endotracheal tube was connected by means of a polythene catheter to a U-tube containing water. This acted as a safety valve and also as a measure of the inflation pressure. Any increase in the end inspiratory pressure being taken generally as an indication for a further dose of curare. The expired gas was passed through a calibrated dry gas meter and away into the atmosphere through a two-way tap (FIG. 1).

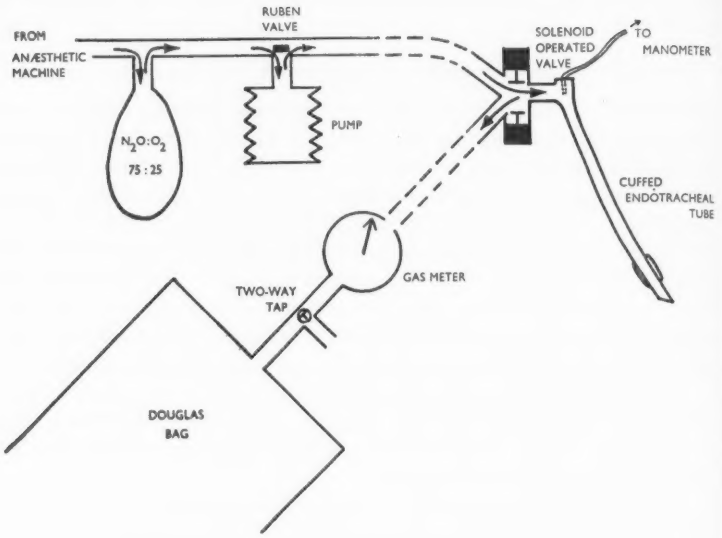

FIG. 1. Arrangement of equipment for estimation of physiological dead space

Expired gas and arterial blood sampling was similar to the procedure carried out pre-operatively. No estimation was carried out within ten minutes of the patient being placed on the operating table. The physiological dead space was measured at the beginning and end of operation, the position of the patient being unchanged between measurements. The body temperature was taken in the oro-pharynx with a mercury bulb thermometer each time the arterial blood was sampled.

Blood and gas analysis

Ten ml of arterial blood were taken into a well lubricated syringe, the dead space of which had been filled with 1:1000 heparin solution. The syringe was capped and immediately placed on ice, the analysis being carried out as soon as possible. The blood was centrifuged at room temperature within the syringe at 1500rpm for five minutes. The P_{CO_2} of the plasma was determined indirectly from the Henderson–Hasselbalch equation. The mean error of this method of determination had previously been determined by tonometer experiments and found to be —0.04mm Hg (SD 1.3) (SE mean 0.36). (FIG. 2). The arterial pH was measured anærobically at 38°C with a glass electrode (radiometer) using the apparatus described by Astrup[9]. The electrode was calibrated

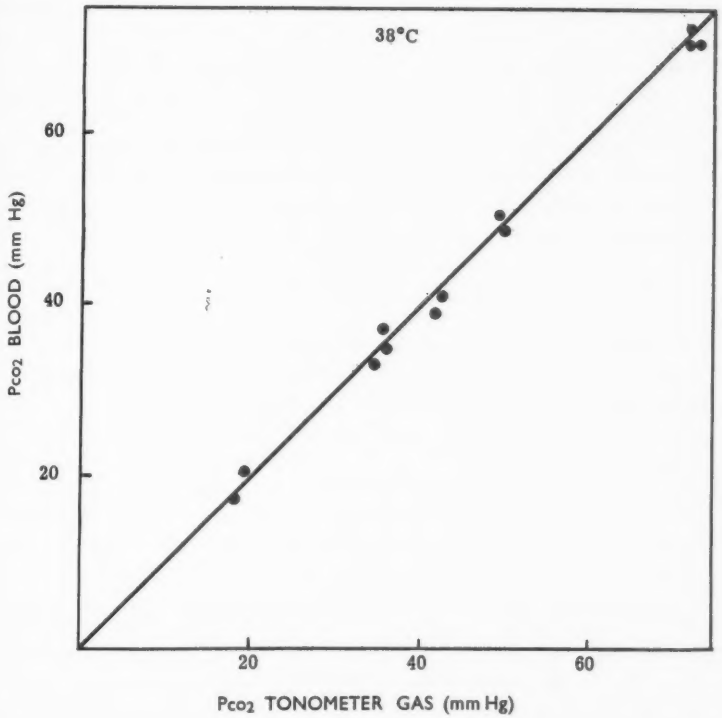

FIG. 2. Carbon dioxide tension of tonometer gas plotted against carbon dioxide tension of blood as derived from the Henderson–Hasselbalch equation

each day at 38°C between pH 4.025 and 6.835 using Hitchcock Taylor buffers. The pH of the blood was compared with the 6.835 buffer. The pK′ for this system had previously been determined by a series of tonometric equilibrations of blood with known tensions of carbon dioxide. (FIG. 3).

Two, 1ml samples of plasma were estimated for CO_2 content using the Van Slyke manometric apparatus. Correction for dissolved nitrous oxide was made using the method of Goldstein and Gibbon[10].

The P_{CO_2} of the blood was derived from the following equation :

$$P_{CO_2} = \frac{\text{Plasma } CO_2 \text{ content m.moles per litre}}{S\,(\text{antilog pH}-pK')+1}$$

where S is the solubility factor for CO_2 in plasma expressed in milli-equivalents per litre mm Hg S=0.0301.

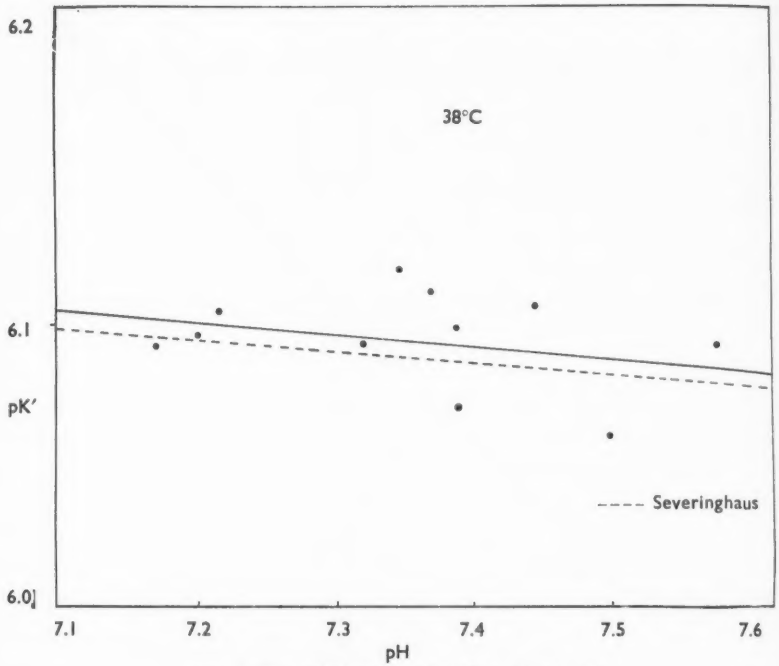

FIG. 3. The relationship of pK′ to pH at 38°C

The P_{CO_2} derived from the above equation was corrected to the temperature of the subject at the time of sampling using the nomogram of Severinghaus[11].

The expired gas was analysed for carbon dioxide content as soon as possible, using the Haldane gas analysis apparatus which had been previously modified for the analysis of carbon dioxide in the presence of nitrous oxide as described by Nunn[12]. All volumes were corected to BTPS. The physiological dead space was derived from the Bohr equation as described above.

Estimation of the anatomical dead space

In six patients breathing air the anatomical dead space was measured using the method of single breath analysis as described by Fowler[6]. These patients were an entirely different group to those in whom the physiological dead space was measured. The patients were premedicated before the estimation was carried out. The anatomical dead space was measured before induction of anæsthesia and again after induction and intubation. The patients were ventilated artificially during anæsthesia using air. All estimations were carried out with the patient

lying supine the head supported by one pillow. Anæsthesia was maintained using intermittent d-tubocurarine, pethidine and thiopentone intravenously, an endotracheal tube having been introduced under suxamethonium relaxation. The inspired and expired gases were sampled continuously by means of a needle valve placed near the endotracheal tube. The sampled gas was passed at the rate of 20ml per minute through the discharge tube of a nitrogen meter (Lundin–Åkesson)[13] at a vacuum pressure of 2mm Hg with a discharge current of 3.5 milli-amperes. The nitrogen meter was calibrated daily from cylinders containing known concentrations of nitrogen. Flow rate was measured with a Fleisch[14] pneumotachograph, which was linear up to 100 litres per minute.

The output of the pneumotachograph and the nitrogen meter was fed into a NEP ultra-violet recorder. After correcting for instrumental lag the anatomical dead space was calculated by geometric estimation from the nitrogen concentration and flow-rate traces. Alveolar nitrogen non-uniformity was measured between 750cc and 1250cc of expired gas. The latter procedure was straightforward where the patient was conscious and able forcibly to expire this quantity of gas from the lungs. However, this was not so during anæsthesia and it was necessary to fill the lungs with 1500cc of oxygen in order to get an adequate quantity of expirate to measure the difference in nitrogen concentration between 750 and 1250cc. During estimation of the dead space the tidal volume was maintained at 750cc, the breath holding time as near to zero as possible and the expiratory flow rate in the region of 75 litres per minute in order to minimise the variations from alterations in these factors. As non-uniformity of alveolar nitrogen would give rise to errors in estimating the anatomical dead space, patients with non-uniformity less than 2.5 per cent were selected for the estimation of the anatomical dead space.

RESULTS

TABLE 1 shows the nature of operation, the position of the patient on the table and the duration of time between samples. Where the position of the patient was head down the degree of tilt was kept the same in all cases at 10° by means of a Pitkin's Tiltometer. The average age of patients was 62 years. The youngest patient was 48 years.

TABLE 2 shows the arterial P_{CO_2}, the tidal volume and respiratory rate. Even though the patients were at rest in bed during the pre-operative estimation of the dead space the arterial P_{CO_2} was on the low side of normal. There is no doubt that some of these patients were hyperventilating to some degree, perhaps as a result of having rather an unpleasant procedure carried out.

Table 2

Arterial P_{CO_2}, tidal volume and frequency of respiration

CASE NO	ARTERIAL P_{CO_2} MM HG PRE-OP.	OPERATIVE		TIDAL VOLUME ML PRE-OP.	OPERATIVE		RESPIRATION/MIN PRE-OP.	OPERATIVE	
1	—	33.5	28.2	—	502	502	—	12	12
2	37.4	30.8	38.4	563	402	402	9	16	16
3	—	28.3	28.3	—	450	450	—	16	16
4	—	31.3	32.2	—	494	494	—	17	17
5	33.5	26.2	34.5	511	450	450	21	16	16
6	—	29.4	27.1	—	484	484	—	14	14
7	34.0	29.5	36.4	401	454	454	27	18	18
8	35.2	32.8	32.8	308	410	410	18	17	17
9	33.4	26.7	27.0	482	392	392	20	16	16
10	—	31.1	29.5	—	379	379	—	17	17
11	—	42.3	36.6	—	388	388	—	21	21
12	—	32.2	27.5	—	440	440	—	18	18
13	—	43.7	48.8	—	441	441	—	18	18
14	—	36.8	37.0	—	443	443	—	13	13

TABLE 3 shows the ratio of V_D/V_T expressed as a percentage, (physiol)
together with the percentage change.

Table 3

Ratio of physiological dead space to tidal volume and percentage change occurring during operation

CASE NO	PRE-OP. % V_D/V_T (physiol)	OPERATIVE % V_D/V_T	% V_D/V_T	%CHANGE
1	—	29.6	28.4	− 4.5
2	43.3	44.1	59.0	+33.7
3	—	49.5	55.8	+12.7
4	—	39.3	38.2	− 2.8
5	36.1	29.2	48.7	+66.7
6	—	34.1	38.4	+12.6
7	55.0	52.1	47.2	− 9.4
*8	45.6	62.7	44.0	−29.8
9	25.7	29.9	33.0	+10.4
10	—	37.2	40.3	+ 8.3
11	—	40.8	56.1	+37.4
12	—	30.2	47.2	+56.4
13	—	32.2	48.8	+ 5.2
14	—	44.6	52.5	+17.7

Mean increase +6% $p = < 0.05$ n=14
Mean increase +7.8% $p = < 0.01$ n=13 (*Exclusion of 8)

Physiological Dead Space ml

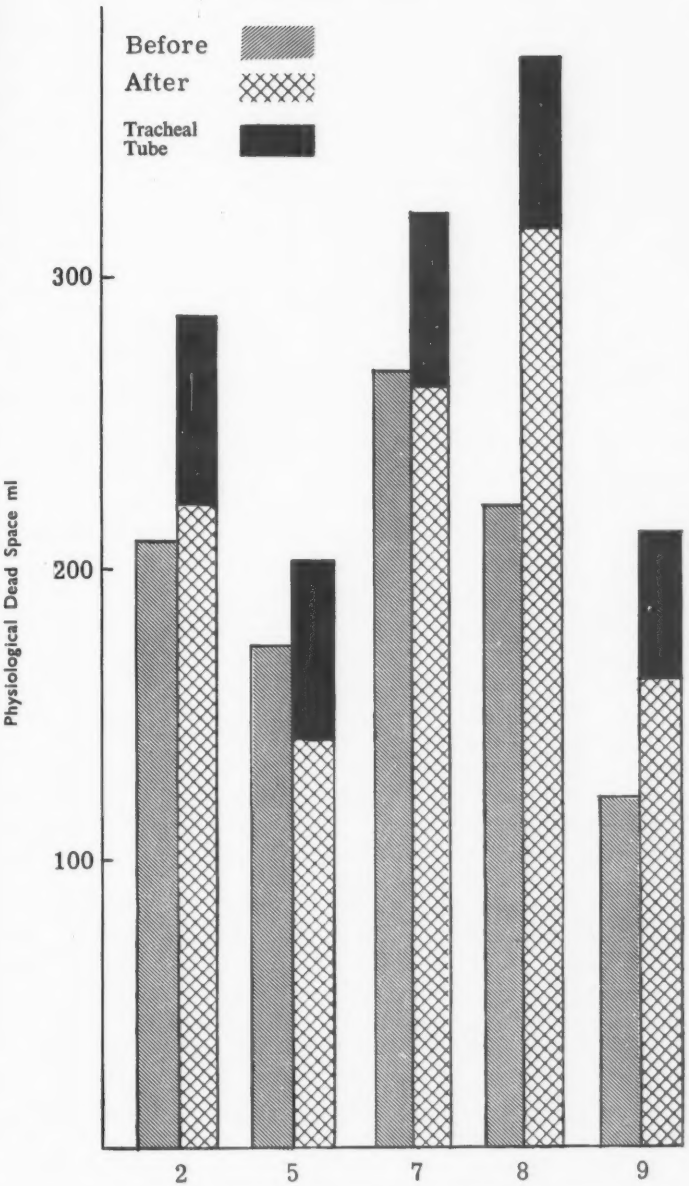

FIG. 4. Physiological dead space before and after induction of anæsthesia and intubation

TABLE 4 shows the physiological dead space volumes in 5 cases

Table 4

Physiological dead space before and after induction of anæsthesia, corrected for a tidal volume of 500ml

CASE NO	PRE-OP. VD MIL. (physiol)	OPERATIVE VD MIL	CORRECTED FOR TUBE
2	216–9=207	221	281
5	181–9=172	141	201
7	275–9=264	260	320
8	228–9=219	312	372
9	128–9=119	150	210

Dead space Ruben valve=9ml

VT=500ml

before operation and at the commencement of operation corrected to 500ml tidal volume and for endotracheal tube and apparatus dead space.

FIG. 4 shows the values in the physiological dead space before and after induction of anæsthesia.

FIG. 5 shows the changes in anatomical dead space following induction and intubation, correction having been made for instrumental dead space.

DISCUSSION

It will be noted that most of the patients display ratios of physiological dead space to tidal volume of more than 30 per cent. This at first sight may appear surprising. However most of the patients were over 50 years of age and in this age group some degree of pulmonary emphysema is to be expected and as a result ventilation-perfusion abnormalities are likely to be present. Several of the patients had their timed vital capacity measured and in most of them the ratio of the one second forced expiratory volume to the forced vital capacity was below 70 per cent, suggesting emphysematous change.

It will also be seen that despite the fact that the tidal volume has been kept constant during the course of operation there is a change in the size of the physiological dead space. If case 8 is included in the series the mean change is + 6 per cent ($P = <0.05$). However, the change of dead space where case 8 is concerned is somewhat anomalous. The figure of 62.7 per cent of tidal volume is abnormally high and the fall (−29.8 per cent) also excessive. There could be two possible explanations for this change. The more likely, that there is an error in the estimation of the physiological dead space which has given rise to this high figure. Secondly, the fall could have been caused by some obstruction, either of a lobar or segmental bronchus. If this had occurred it would almost certainly have been reflected by an increase in the inflation pressure (this was not noted). There was no clinical evidence

of obstruction and no post-operative pulmonary complication. If we exclude case 8 on the grounds of experimental error then it would appear that the mean increase which is now 7.8 per cent is highly significant ($P = <0.01$).

One might have expected the increase in the physiological dead space to be related to time but this was not possible to demonstrate. Neither was it possible to show any relationship between posture and these changes.

The physiological dead space can be taken as an index of effective alveolar perfusion. In health ventilation and perfusion closely balance one another ($V_A/Q_C=0.8$). During anæsthesia and in disease this is not always the case. Campbell and Nunn[1] have shown that there is an increase in the physiological dead space when a patient under controlled artificial ventilation during general anæsthesia is compared with the same patient not under an anæsthetic and breathing spontaneously.

The findings in the five cases studied pre-operatively in the present series agree with these findings, *i.e.* volume for volume artificial ventilation is less effective than spontaneous ventilation and that intubation, by cutting down the anatomical dead space, helps to compensate for this change.

The mean increase in the physiological dead space comparing pre-operative and operative measurements in this series was 77ml (after correction for endotracheal tube 70ml). The mean increase was 20ml if no correction was made for endotracheal tube. The mean increase as measured by Campbell *et al*[1] was 62.8ml.

The mean decrease in the anatomical dead space after the passage of an endotracheal tube was 71ml (FIG. 5). There was no demonstrable change in the alveolar nitrogen non-uniformity during the course of operation compared with the pre-operative figure in *normal subjects*, neither was there a significant change in the anatomical dead space in *normal subjects* during the course of operation. It is important to stress that these were normal subjects as these findings may not necessarily obtain to the patients in whom the physiological dead space was studied. This latter group must be considered to be suffering from some degree of pulmonary emphysema. This is a common condition in which uneven ventilation and distribution of gases occurs and it is likely to be accentuated by artificial ventilation, the very mechanics of which encourage maldistribution in the diseased lung.

If one makes the assumption that there has been no change in the non-uniformity of distribution of gases and no change in the anatomical dead space in the group in which the physiological dead space was studied, then the variations in physiological dead space must be due to alteration in the distribution of perfusion.

How could the changes in distribution of pulmonary blood flow be

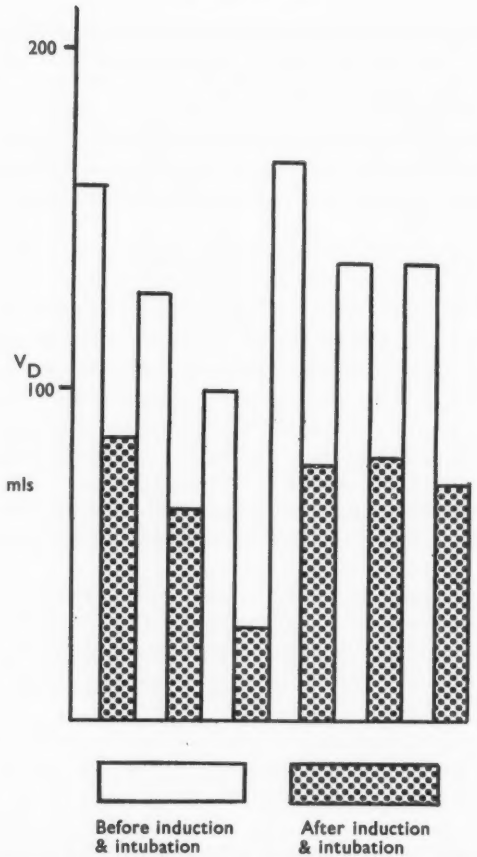

FIG. 5. Anatomical dead space before induction and intubation and after

brought about? They could result from a fall in cardiac output but under light anæsthesia with nitrous oxide, a fall in cardiac output does not usually occur[15]. In only one case in the present series was there a marked fall in blood pressure towards the end of operation, *i.e.* circumstances in which the cardiac output might have fallen.

An increase in inflation pressure might give rise to (*a*) a fall in cardiac output resulting from reduction of venous return or (*b*) an alteration in the distribution of perfusion. Howell *et al*[16] have shown that in order to maintain a constant volume in the pulmonary vascular bed, an increase in alveolar pressure calls for an increase in perfusion pressure. The likely reasons for an increase in inflation pressure would be the waning of the effect of the d-tubocurarine or some obstruction in the air passages but an increased inflation pressure was not observed.

The effect of posture on ventilation-perfusion relationships within the lungs has been studied by Svanberg[17] and by Martin and Young[18], Bjorkmann[19] and Mateson and Carlens[20] who demonstrated gravitational redistribution of blood following change in posture. Comparable changes may occur in the paralysed subject during general anæsthesia.

Further studies are being carried out to ascertain the extent to which these changes occur during anæsthesia in patients with normal lungs.

SUMMARY

Physiological dead space was measured in fourteen anæsthetised patients under controlled artificial ventilation. There was a mean increase of 6 per cent during the course of operation. The possible reasons for the increase have been discussed. In five cases the physiological dead space was estimated pre-operatively and compared with values obtained during anæsthesia. The anatomical dead space has been measured before and after induction of anæsthesia using the method of Fowler. The non-uniformity of alveolar nitrogen has been followed during the course of operation.

Acknowledgements

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References

[1] CAMPBELL, E. J. M., NUNN, J. F. and PECKETT, B. W. (1958). *Brit. Anæs.*, *30*, 166.
[2] HOWELL, J. B. L. and PECKETT, B. W. (1957). *J. Physiol.*, *136*, 1.
[3] BOHR, C. (1909). *Skandinav. Arch. f. Physiol.*, *22*, 221.
[4] PAPPENHEIMER, J. R. (1950). *Fed. Proc.*, *9*, 602.
[5] ENGHOFF, H. (1938). *Upsala lak. foren. forh.*, *44*, 191.
[6] RILEY, R. L. and COURNAND, A. J. (1949). *J. appl. Physiol.*, *1*, 825.
[7] FOWLER, W. S. (1948). *Amer. J. Physiol.*, *154*, 405.
[8] HILL, D. W. and HOOK, J. R. (1958). *J. Physiol.*, *143*, 1.
[9] ASTRUP, P. (1956). *Scandinav. J. Clin. & Lab. Invest.*, *8*, 33.
[10] GOLDSTEIN, F., GIBBON, J. H., ALLBRITTEN, F. F. and STAYMAN, J. W. (1950). *J. Biol. Chem.*, *182*, 815.
[11] SEVERINGHAUS, J. W., STUPFEL, M. and BRADLEY, A. F. (1956). *J. appl. Physiol.*, *9*, 201.
[12] NUNN, J. F. (1958). *Brit. J. Anæs.*, *30*, 254.
[13] LUNDIN, G. and AKESSON, L. (1954). *Scand. J. Clin. & Lab. Invest.*, *6*, 250.
[14] FLEISCH, A. (1956). *Helv. Physiol. Acta.*, *14*, 363–368.
[15] PRIME, F. J. and GRAY, T. C. (1952). *Brit. J. Anæs.*, *24*, 101.
[16] HOWELL, J. B. L., PERMUTT, S. and PROCTOR, D. F. (1958). Annual Meeting Amer. Phys. Soc. Philadelphia.
[17] SVANBERG. (1957). *Scand. J. Clin. & Lab. Invest.*, *9*, Suppl. *25*, 1.
[18] MARTIN, C. J. and YOUNG, A. C. (1957). *J. appl. Physiol.*, *II*, 371.
[19] BJORKMANN, S. (1934). *Acta Med. Scandinav. Suppl.*, *56*.
[20] MATESON, S. B. and CARLENS, E. (1955). *J. Thoracic Surg.*, *30*, 676.

Prolonged intubation in neonatal tetanus

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Despite the use of the recent phenothiazine derivatives the mortality rate in neonatal tetanus remains high. Wright[1] quotes an overall mortality rate of 82.5 per cent in a consecutive series of 217 neonates treated by various forms of sedation. Adams *et al* [2] report a mortality rate of 95 per cent in severe cases treated with chlorpromazine. They feel that in many cases death is due to respiratory failure probably resulting from tetanus intoxication of the medullary centres. Therefore attempts are now being made to treat severe cases by inducing total paralysis with a muscle relaxant and maintaining the ventilation mechanically[3 4]. Although prolonged intubation has been utilised in adults[5 6 7], it is generally accepted that owing to the risk of laryngeal damage[8], a tracheostomy should be performed if ventilation is to be continued for more than twenty-four hours. The difficulties of managing a tracheostomy in a neonate are, however, considerable. The head is large, the neck relatively short and the trachea narrow. Difficulties in the period of weaning from the tube are also common[3]. A further trial of prolonged endotracheal intubation as an alternative to tracheostomy was therefore considered justifiable. The results are detailed in the case histories which follow.

CASE NO 1

African male, born 24th May 1959 and admitted 29th May 1959, with severe spontaneous reflex spasms. Incubation period – four days. Period of onset – nine hours. The patient was sedated with sodium phenobarbitone (total 200mg) and acetylpromazine (notensil) (total 50mg) but after twenty hours respiratory failure occurred. It was characterised by shallow, gasping respirations at a rate of 4 per minute.

At 6 pm on 30th May 1959, 20mg of suxamethonium was given intramuscularly (IM) and endotracheal intubation was performed with a size 00 flexometallic tube. Paralysis was maintained with curare in doses of 3mg IM and ventilation was effected with a Radcliffe Mark V positive pressure respirator. On 3rd June the larynx was inspected and a similar tube replaced. The whole larynx was œdematous and the cords were not visible. No ulceration was seen. On the 6th, 8th and 9th June the tube was accidentally removed. The nurse on each occasion used a face-mask to

inflate the lungs until the tube could be re-inserted and on each occasion regurgitated stomach contents were forced into the trachea. These were subsequently removed by a suction catheter passed down the endotracheal tube. A marked air leak was noted on 9th June. Inspection revealed that the larynx was dilated. The probable cause was pressure from the shoulder of the flexometallic tube. A size 0 latex armoured tube was used as a replacement.

On the 10th June bronchoscopy was performed. The vocal cords were seen as thin white lines in the œdematous vocal folds. The epiglottis was less œdematous. There was a white circumferential slough in the cricoid region which was firmly attached to the tracheal wall. A size 1 latex armoured tube was inserted easily. A size 2 tube was inserted on the 12th and a size 3 on the 20th June: on each occasion the leak had become too great to allow effective ventilation.

By 23rd June the patient was breathing spontaneously during the day though still on the respirator at night. An attack of gastro-enteritis at this time so weakened the patient that he had to be maintained completely on

FIG. 1 Sagittal section of trachea from case 1. The dilated portion of the trachea is lined by sloughs. The lower portion of the trachea and the main bronchi are of normal size.

the respirator. Bronchoscopy was again performed on 25th June. Œdema at the base of the tongue was such that the tip of the epiglottis only projected 5mm from the glosso-epiglottic fold. The laryngeal orifice had an antero-posterior diameter of 8–9mm with a marked groove posteriorly. There was no visible tracheo-œsophageal fistula. The trachea was grossly dilated and covered with sloughs, but the carina and bronchi were of normal size. Pus was exuding from both main bronchi.

Death occurred on 7th July 1959 from bronchopneumonia.

At autopsy there was a confluent bronchopneumonia involving all portions of the lung. The larynx and upper two thirds of the trachea were grossly dilated (FIG. 1), and the epiglottis, base of tongue and larynx markedly œdematous (FIG. 2). Sloughs lined the whole of the interior of the larynx and the upper part of the trachea.

FIG. 2 Posterior view of specimen from case 1, showing soft palate and grossly dilated laryngeal orifice. The folds in the arytenoid region are due to the marked œdema which was present in the fresh specimen

COMMENT

It proved extremely difficult to devise a satisfactory form of fixation for the endotracheal tube. Numerous types of harness were tried but all were either ineffectual or else resulted in pressure necrosis of the lips, face or fauces. At one period the catheter mount created a depression in the frontal bones 1cm deep and at another an elastic harness (similar in principle to that of Hudson) resulted in overriding of the cranial bones. Despite the frequency with which the tube was removed and aspiration of gastric contents occurred, immediate endotracheal suction and vigorous physiotherapy proved remarkably effective in preventing chest complications.

The interesting feature of the case was the progressive dilation of larynx and trachea. This could have been due to the backward pressure exerted by the spring in the tube, to the dilator effect of the shoulder on the earlier tubes or to the pressure wave of the advancing gases. It is unlikely that the latter was responsible as the dilation ceased abruptly just above the carina.

FIG. 3 Posterior view of larynx and epiglottis of case 2 to show œdema and ulceration of tip of epiglottis (fixed specimen)

CASE NO 2

African female born 26th August 1959, and admitted 31st August with almost continuous severe spasms. Incubation period – four days. Period of onset–six hours. Temperature: 102°F (38.8°C) on admission. It was only after adequate air entry had been achieved by intubation under relaxation with a size 00 flexometallic tube and artificial ventilation that diffuse crepitations were heard, thus confirming the previous suspicion of bronchopneumonia. Procaine penicillin 300,000 units was given twice daily and continued until death. The pneumonia improved but on the fourth day of treatment, the endotracheal tube was accidentally removed. Severe anoxia occurred and gastric contents were aspirated. Laryngoscopy at this time revealed marked œdema but no ulceration or sloughing. A size 00 latex armoured tube was inserted and left in place for the duration of the illness. Relaxants were withdrawn and sedation was commenced on the sixth day but complete control of spasms was not achieved until the eighth day. By the fifteenth day weaning from the respirator was complete but on the

FIG. 4 Sagittal section of trachea and larynx from case 2. Sloughs lined larynx and trachea. The bulge below the tracheostomy opening can be clearly seen

sixteenth day pneumonia recurred. Streptomycin 0.25g daily was added to the penicillin. On the nineteenth day an attempt to remove the endotracheal tube was unsuccessful. The larynx was œdematous and distorted and a pressure ulcer was present on the right side of the lower lip. The presence of sloughs in the trachea prevented subsequent extubation. A tracheostomy was accordingly performed on the twenty-fifth day. However, the tracheostomy tube became repeatedly blocked with slough and the baby died on the twenty-sixth day. Post-mortem examination revealed a diffuse bronchopneumonia with pus in both main bronchi. The epiglottis, base of tongue and larnyx were œdematous and the tip of the epiglottis was ulcerated (FIG. 3). The larynx and trachea were ulcerated and sloughs were present (FIG. 4). Dilation of larynx and trachea was not as marked as in case 1 but there was a posterior bulge in the trachea, where pressure had been exerted by the tracheostomy tube (FIG. 4).

DISCUSSION

Despite earlier fears tracheostomy in the neonate has proved to be safer and easier to manage than prolonged oral endotracheal intubation. This is probably due to the extreme difficulty of fixing an endotracheal tube in such a manner that it will not be displaced during the execution of common nursing procedures and physiotherapy. The maxilla is the only practicable anchorage but does not lend itself easily to this purpose. Even if difficulties of fixation could be surmounted, it is doubtful whether weaning from the tube could be effected without temporary tracheostomy in some cases.

Although œdema was the only abnormality observed at the first laryngoscopy on both these cases, it is well known that the sub-glottic component may produce a fatal outcome[9]. Therefore it seems unwise to continue endotracheal intubation in neonates and young children for periods of more than 12–24 hours.

SUMMARY

Two cases of prolonged intubation associated with the use of intermittent positive pressure respiration in neonatal tetanus are described.

It is concluded that tracheostomy is both safer and easier to manage, even in the neonate.

Acknowledgements

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References

[1]WRIGHT, R. (1960). *S. Afr. med. J.*, *34*, 111.

[2]ADAMS, E. B., WRIGHT, R., BERMAN, E. and LAURENCE, D. R. (1959). *Lancet*, *1*, 755.

[3]SMYTHE, P. M. and BULL, A. (1959). *Brit. med. J.*, *2*, 107.
[4]SYKES, M. K. (1960). Intermittent positive pressure respiration in tetanus neonatorum. *Anæsthesia*, *15*, 4, 401.
[5]FOREGGER, R. (1946). *Anesthesiology*, *7*, 285.
[6]BRIGGS, B. D. (1950). *Anesthesiology*, *11*, 129.
[7]URRY, A. G. (1951). *Anesthesiology*, *12*, 662.
[8]ANNOTATION. (1955). *Lancet*, *2*, 79.
[9]HAINS, A. M. and POWELL, K. J. (1955). *Brit. J. Anæsth.*, *27*, 257.

Intermittent positive pressure respiration

In tetanus neonatorum

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Tetanus neonatorum occurs rarely in most European countries. In Great Britain there were only thirty-six deaths from this cause during the period 1938–47[1]. The incidence, however, is higher in certain overseas centres. Large series have recently been reported from Singapore[2], Ibadan[3 4] and Brazil[5] whilst the disease is not uncommon in the U.S.A.[6 7 8] and there is one report from China[9]. In South Africa the disease is reported to occur most frequently amongst the coloured population, but it is suspected that many cases occurring in Africans are not officially recorded[10]. There are several recent reports from South Africa[11 12 13 14 15] which suggest that the highest incidence is in Durban. Wright[16] reports that 246 cases were admitted to the King Edward VIII Hospital, Durban, between May 1956 and April 1959. The ratio of African to Indian babies admitted was 10:1 despite the equality of the populations at risk. Although a combination of chlorpromazine with barbiturates proved to be a most effective method for controlling spasms, there was no significant difference in the mortality rate when different sedative régimes were subjected to controlled trials. The overall mortality rate during this three-year period was 82.5 per cent, but of sixty-four cases having spontaneous reflex spasms on admission (the most useful criterion of severity) only one survived. The poor response to sedative drugs has prompted further therapeutic trials. This paper deals with the technical aspects of one such study – the use of muscle relaxants and intermittent positive pressure respiration (IPPR).

CAUSES OF DEATH

The exact mechanism of death in tetanus neonatorum is often difficult to determine. There appear to be four main groups:

1 Uncontrolled spasms – usually terminating within forty-eight hours
2 Respiratory failure – occurring between the third and fourteenth day
3 Marasmus and terminal bronchopneumonia between the third and sixth weeks
4 A miscellaneous group which includes aspiration of feeds, ulceration of the œsophagus and peritonitis.

It would seem at first sight that total paralysis and intermittent positive pressure respiration might prevent most of those complications. Spasms can be controlled, the airway and ventilation assured and chest complications treated effectively. Unfortunately, the technique is not without risk and its application to neonates introduces further hazards. Moreover, the longer survival time of cases treated by this method reveals a further stage in the pathology of the disease, namely, circulatory failure. This may be due either to toxic myocarditis[17] or to medullary involvement. The recent history of the treatment of bulbar poliomyelitis provides a close parallel. Death from respiratory failure has become rare but may be replaced at a later date by death from circulatory failure. At the time of writing, relatively little is known about the pathological and physiological changes involved; as a result recovery from circulatory failure is infrequent.

Support for the suggested medullary origin of the respiratory and circulatory failure is provided by Baker[18] who has demonstrated pathological changes in the medulla of adult cases who have died from severe tetanus. He noted that the vagus nucleus was particularly involved and that the onset of gasping respiration or apnœa, which may occur as long as forty-eight hours after the last dose of sedative drug, is nearly always accompanied by unconsciousness and generalised flaccidity. A similar state may be produced in the experimental animal by the injection of large doses (over 20MLD) of tetanus toxin[19]. The above evidence does not, of course, exclude toxic myocarditis as a possible cause of death in some of the cases terminating in circulatory failure.

Despite these possible drawbacks, it seems logical to provide mechanical assistance for the respiration whilst respiratory inadequacy exists. Such a régime has been described by Smythe and Bull[20]. However, in a busy general ward, respiratory failure of sudden onset may not be recognised until it is too late to institute effective treatment. To

overcome this difficulty and to ensure standardisation in a controlled trial, it was decided that total paralysis and artificial respiration should be commenced as soon as the severity of the disease had been assessed. Similarly, conservative treatment was instituted in the control group of cases at the earliest possible moment. The experience gained in the early stages of this study forms the basis for the following observations. The results of the tetanus trials will be reported elsewhere at a later date.

TECHNIQUE

For the purpose of the trial this was standardised as follows: Intubation was performed as soon as possible after admission to the IPPR unit. Oxygen was administered by face mask and 20mg of suxamethonium was then given intramuscularly. When apnœa occurred a laryngoscope was passed, the pharynx was cleared by suction and a size 00 flexometallic tube was passed into the larynx. A soft plastic tube was also passed into the stomach for feeding purposes. The patient was connected to a mechanical ventilator and the machine adjusted until the tidal volume and rate appeared to be adequate. When spontaneous respiration or tetanic spasms returned, 3mg of d-tubocurarine was injected intramuscularly. This was repeated whenever twitchings or respiration reappeared. Tidal volume was subsequently adjusted on the basis of measurements of arterial P_{CO_2}, obtained by a rebreathing method[21]. Additional oxygen was only used if cyanosis occurred.

An arbitrary limit of twenty-four hours from intubation was set for tracheostomy, though in most cases this was performed within twelve hours. The thyroid isthmus was retracted or divided and a window made in the front of the trachea at the level of the second and third rings. A cut-down size 00 latex armoured endotracheal tube was passed through this for a distance of 1cm.

The optimal duration of artificial respiration has not yet been determined. In surviving cases of severe neonatal tetanus, treated by conservative methods, spasms rarely persist for more than two or three weeks. However, in one case treated by IPPR spasms were still present four weeks after the onset of the disease. Residual stiffness has persisted in some cases for as long as three months. In the neonate a marked reduction in relaxant requirements, indicative of recovery, does not appear to occur. One is therefore forced to substitute sedative drugs for the relaxant at intervals until the spasms can be adequately controlled by the drug employed. Only then can weaning from the respirator be attempted, progress in this respect being judged solely by the maintenance of an adequate tidal volume.

Feeding is accomplished in most cases by gastric tube. Half strength milk is used initially and when this is well tolerated full strength expres-

sed-breast milk is given, the volume being calculated on the basis of surface area. Aspiration of gastric contents is performed before each feed. The volume is measured and the aspirate is returned as the initial part of each feed. A full feed is only given if the aspirate is less than one-quarter of the previous feed, a half-feed is given if the aspirate is between a quarter and a half of the previous feed and no feed at all if it exceeds half the previous feed. In this way a continual check is kept on gastric emptying.

Retention of urine is generally no problem, but catheterisation may be necessary for one or two days.

The latex armoured tracheostomy tube is replaced by a metal Alder Hey tracheostomy tube[22] when it is certain that the respirator will no longer be required. Weaning from this tube is not attempted until all the feeds are being taken by mouth. Progress to this stage is often slow and much patient nursing is required to achieve success. A medicine dropper and later a 'Belcroy' feeder are helpful in establishing swallowing. When this has been accomplished the tracheostomy tube occluder is inserted for increasing periods. The tube is removed when the occluder can be tolerated for twenty-four hours and the stoma is kept open by a rubber plug. This is, in turn, removed and a dressing applied until the stoma closes.

APPARATUS

1 *Respirator*

Certain modifications are required to the apparatus commonly used for adults. The tidal volume of a neonate breathing spontaneously at 35–40 respirations per minute is normally about 16ml. This small volume is insufficient to actuate present commercial flutter valves, unless the device originated by Bull is utilised[20]. Mechanically operated valves overcome this difficulty and enable the dead space to be minimised by the separation of inspiratory and expiratory gas streams at the junction of delivery tubing and tracheostomy tube. A connection such as that shown in FIGS. 1 and 2 has been found of value. The Y-piece is locked to the horizontal limb of the connection on the tracheostomy tube by a half turn and can be interchanged rapidly with an oxygen supply unit for emergency purposes.

Both Radcliffe Mark V (positive pressure) and Smith-Clarke (positive/negative) pumps have been found satisfactory. The air should either be led in from outside the building or else passed through a bacterial filter of non-absorbent cotton wool situated well off the floor. The filter should have a large cross-sectional area to minimise resistance to flow and the depth of wool should be at least two inches (5cm). The wool should be changed daily.

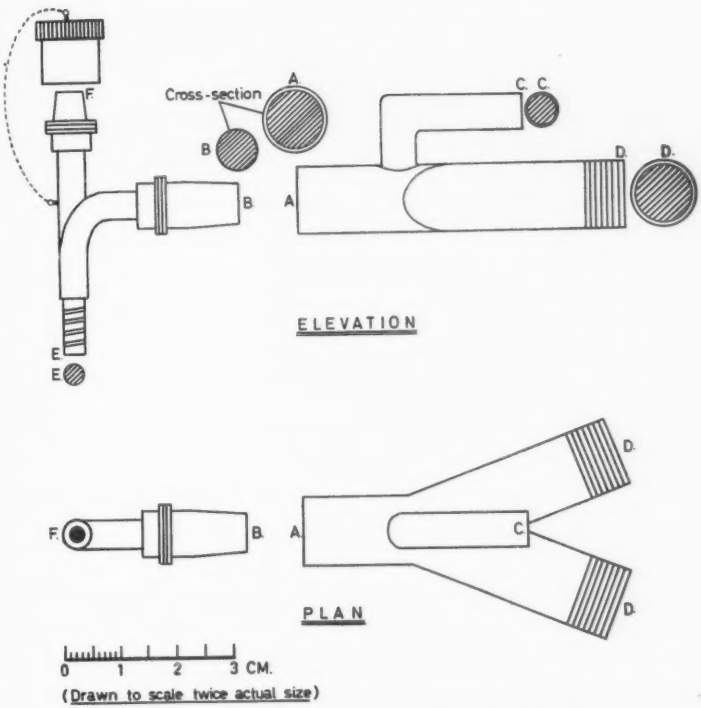

FIG. 1 Diagram of tracheostomy tube suction connection shown in fig. 2

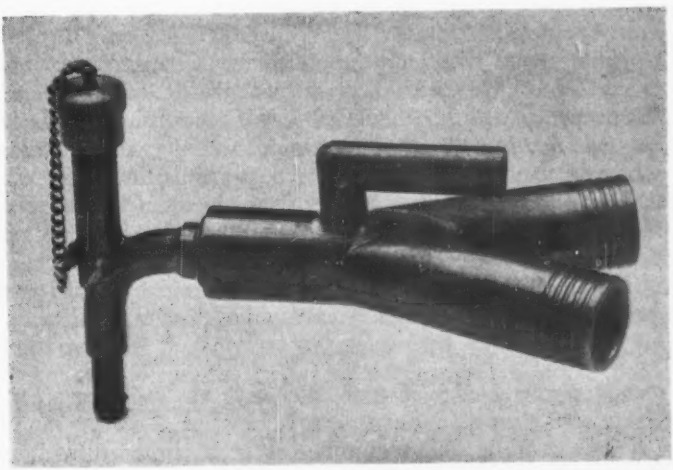

FIG. 2 Tracheostomy tube suction connection and Y–piece for respirator tubing. The small right-angled tube is for the connection to the manometer

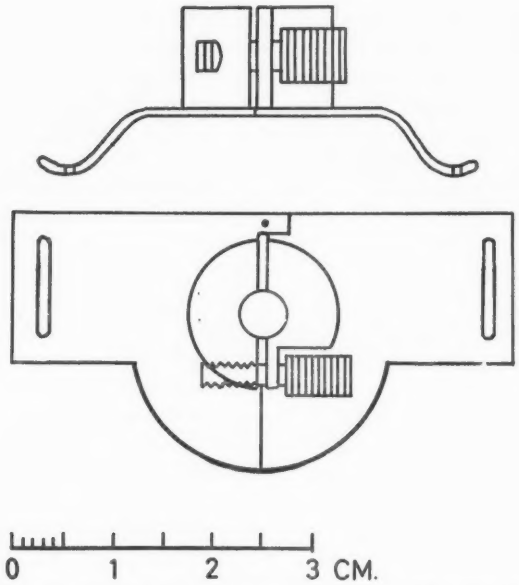

FIG. 3 Tracheostomy tube holder. Made in three sizes with internal diameter of 4.5 mm, 5.5 mm and 7 mm to suit tube sizes 00, 0 and 1 respectively

2 *Humidifier*

When small minute volumes are delivered the passage of warm moist air from humidifier to patient is slowed. Cooling is therefore more marked and unless the water temperature is set at a higher level (about 150°F or 71°C when using a 4ft or 120cm plastic delivery tube), the gas may not be adequately humidified. This procedure, however, is not without danger for temporary disconnection of the patient allows a much greater volume of air to flow through the humidifier. (This applies particularly to the Radcliffe Mark V pump where the bellows will fall the full distance). On reconnection, therefore, the patient may be subjected to air at a dangerously high temperature for several breaths. The only remedy is to improve lagging on the delivery tube or to shorten the latter to the minimum by hanging the humidifier on to the side of the cot. In this connection a small humidifier of the Spalding type[23] having a surface area of 240cm has been found to ensure 100 per cent humidity when small tidal volumes are involved.

3 *Tracheostomy tubes*

The small diameter of a neonate trachea leaves little room for an inflatable cuff, even on a metal tube. In practice, leakage round the tube is not a problem. Bull uses a 'portex' tube, hardened by dipping

into ether for exactly one minute and then allowing it to dry in the open for twenty-four hours. Shortened latex armoured tubes are satisfactory if carefully handled. Care must be taken that the wire spiral is not kinked during preparation or difficulty may be experienced in passing the soft rubber suction catheter (No 3 EG) which is used for aspirating the trachea. These tracheostomy tubes are made by dividing the standard endotracheal tubes into two with sharp wire cutters or scissors. The protruding end of the wire spiral is extracted from the rubber for a short distance, cut off, and the end which remains is then turned so that it is again embedded in the rubber. A Magill type suction union is inserted into the uncut end. As shown in FIG. 1, the plug occluder for the suction limb is replaced by a cap. This provides greater protection against bacterial contamination. All connections are made by means of a quick action joint utilising a taper and coarse thread. This results in a leakproof joint in which risk of accidental disconnection is reduced to a minimum. The tracheostomy tube is grasped by the adjustable collar on the holder (FIG. 3) and this in turn, is held in place by a harness, made from tape padded with gauze, and surrounded by waterproof strapping for most of its length. One tape passes around the neck and through the holes in the baseplate of the holder. The other stretches from the baseplate, under one axilla, across the back, and up under the other axilla. This strap prevents displacement of the tube in a cephalad direction. Such a harness has been found to be surprisingly effective in preventing accidental dislodgement of the tube.

SUCTION TECHNIQUE

It is essential that the utmost sterility be preserved during this procedure and that it should be performed expeditiously to avoid anoxia. The latter requirement precludes the use of a 'no touch' technique ; in neonates pre-oxygenation is of value when chest complications are present. Suction packs consist of two No 3 EG and one No 7 EG Jacques catheters with tips cut off. The large catheter also has side holes to minimise pharyngeal trauma. A plastic connector and 2ft of latex pressure tubing are included. These articles are wrapped in a sheet of 'portex' plastic 2ft 3in (67.5cm) long × 1ft 3in (37.5cm) wide × 0.003in (0.075mm) thick ; a hole 1cm in diameter is situated in the centre line 10in (25cm) from one end of the sheet. The whole package is autoclaved in a paper bag. In use, the plastic sheet is opened and spread out over the baby so that the hole is approximately over the end of the suction limb of the tracheostomy tube connection. The nurse then scrubs for three minutes with 'phisohex'. She connects the latex tubing to the sucker (holding the 'dirty' connection in a swab) and then cleans the cap and tip of the suction limb, which protrudes through the

sheet, with 1 per cent cetavlon. The cap is removed, the catheter is inserted and suction applied as it is withdrawn. It is thus possible to carry out suction quickly and without introduction of bacterial contamination. The transparency of the sheet allows the patient's colour and chest movements to be observed throughout the procedure.

COMPLICATIONS ENCOUNTERED

1 *Chest complications*

Infection and collapse are common unless strict precautions are observed. The normal protective mechanisms are: filtration and humidification of the air in the nose; the ciliary 'escalator'; movement of the lungs and coughing.

These procedures must be simulated artificially. The inspired air must be free from infective organisms and humidity must be maintained at the optimal level by frequent checks on the temperature of the inspired gas. This can be done by inserting a clean thermometer into a small hole in the inspiratory tube; the temperature should be 90°F (±5°F). Regular turning of the patient helps to prevent pulmonary congestion. Coughing can be simulated by percussion, vibration and by a rapid squeeze of the whole chest after a maximal inspiration. It is helpful to use manual inflation with oxygen to assist this process. As noted above, scrupulous asepsis must also be practised when aspirating secretions. If atelectasis does occur, it can nearly always be cleared by posture or prolonged physiotherapy. A polythene tube can be bent and directed down the left bronchus to facilitate the clearance of secretions on this side. The end should be smoothed by friction and side holes should be pierced to reduce the suction force on the terminal orifice. It should be noted that autopsy specimens have revealed ulceration of the carina after the regular use of such a catheter.

2 *Facial œdema*

This is especially marked in neonatal tetanus treated by conservative or IPPR methods, but it may also occur in other conditions in which the face is rendered immobile. There appears to be no upset of the plasma protein level though there may be sodium and chloride retention in some cases. Strict control over fluid balance is essential, particularly in the early stages.

3 *Hypothermia*

With either form of treatment body temperature tends to drop rapidly when the environmental temperature is 65–75°F. The temperature usually climbs slowly and becomes normal about the tenth day. This marked fall has not been noted during the humid summer months. If the hypothermia is allowed to persist it should be remembered that

the metabolic rate is decreased (7 per cent per °F fall of temperature)[24] and allowance should be made for this when calculating calorific intake and fluid requirements.

4 *Liver enlargement*

Progressive enlargement of the liver has been noted in all cases ventilated with positive pressure alone. Twenty-four hours after the commencement of IPPR the liver may be 3–6cm, below the costal margin, but in no case has the liver descended further than 7cm below the costal margin. Application of a negative phase during expiration will reduce the liver to normal size. If the negative pressure is too marked, however (more than 6cm H_2O), the lung compliance falls so markedly that ventilation may be conspicuously reduced. Measurement of pressures in the inferior vena cava during IPPR with varying inspiratory wave forms and pressures has so far revealed little alteration in the mean venous pressures. It appears that the extent of the pressure swing may be the determining factor in the liver enlargement in neonates. Further work is in progress on this problem.

5 *Circulatory failure*

This appears to occur in two forms. First, there is the peripheral failure of slow onset which inevitably accompanies terminal pneumonia. Second, there occurs an acute circulatory failure without obvious cause. The patient, who is otherwise progressing satisfactorily dies unexpectedly. At autopsy no abnormalities are found which would account for this. Technical causes may be responsible, but the possibility of a toxic myocarditis or medullary involvement must be entertained.

6 *Paralytic ileus*

This usually appears in the terminal stages of an overwhelming chest infection. Minor degrees of abdominal distension are common in the earlier stages of the disease but usually resolve if feeding is withheld for twelve hours.

CONCLUSION

IPPR in the neonate presents several new problems. These are : technical difficulties in the provision of correct ventilation ; the prevention of chest complications ; the management of, and weaning from, the tracheostomy.

The above experiences have been reported not only because of their rather limited application to the field of neonatal tetanus but also because it is believed that there is scope for a wider application of IPPR to other neonatal and infant conditions.

Acknowledgements

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References

[1] CONYBEARE, E. T. and LOGAN, N. P. D. (1951). *Brit. med. J.*, *1*, 504.
[2] LOH SIEW GEK. (1951). *Med. J. Malaya*, *5*, 181.
[3] JELIFFE, D. B. (1950). *Arch. Dis. Childh.*, *25*, 190.
[4] TOMPKINS, A. B. (1958). *Brit. med. J.*, *1*, 1382.
[5] PINHEIRO, D. (1957). *J. Pediat.*, *51*, 171.
[6] VINNARD, R. T. (1945). *Surgery*, *18*, 484.
[7] SPIVEY, O. S., GRULEE, C. F. and HICKMAN, B. T. (1953). *J. Pediat.*, *42*, 345.
[8] AXNICK, N. W. and ALEXANDER, E. R. (1957). *Amer. J. publ. Hlth.*, *47*, 1493.
[9] KAO YUNG ENG. (1951). *Clin. med. J.*, *69*, 512.
[10] SLOME, R. (1954). *S. Afr. med. J.*, *28*, 473.
[11] FRIEDLANDER, F. C. (1951). *J. Pediat.*, *39*, 448.
[12] KLENERMAN, P. and SCRAGG, J. N. (1955). *S. Afr. med. J.*, *29*, 853.
[13] FALCKE, H. C. (1957). *Med. Proc.*, *3*, 171.
[14] LAURENCE, D. R., BERMAN, E., SCRAGG, J. N. and ADAMS, E. B. (1958). *Lancet*, *1*, 987.
[15] ADAMS, E. B., WRIGHT, R., BERMAN, E. and LAURENCE, D. R. (1959). *Lancet*, *1*, 755.
[16] WRIGHT, R. (1960). *S. Afr. med. J.*, *34*, 111.
[17] ALHADY, S. M. A., BOWLER, D. P., REID, H. A. and SCOTT, L. T. (1960). *Brit. med. J.*, *1*, 540.
[18] BAKER, A. D. (1942). *J. Neuropath. exp. Neurol.*, *1*, 394.
[19] BRONNTMANN, R. and STIRNEMANN, H. (1956). Proceedings of the World Congress of Anæsthesiologists, Netherlands, 1955, p.282. Burgess Publishing Co., Minneapolis, U.S.A.
[20] SMYTHE, P. M. and BULL, A. (1959). *Brit. med. J.*, *2*, 107.
[21] SYKES, M. K. (1960). *Anæsthesia*, *15*, 4, 394.
[22] NISBET, H. I. A. and WILSON, F. (1958). *Brit. J. Anæsth.*, *30*, 419.
[23] MARSHALL, J. and SPALDING, J. M. K. (1953). *Lancet*, *2*, 1022.
[24] WRIGHT, SAMSON. Applied Physiology. Oxford University Press, 9th ed., 1952, p.378.

Methohexital

A short acting barbiturate

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Induction of anæsthesia for surgery or for dental extractions is commonly achieved by the use of nitrous oxide or of intravenous thiopentone. The latter with appropriate dosage and rate of administration produces sleep or narcosis. The normal induction dose of 250–500mg of 2.5 per cent thiopentone or the use of repeated smaller doses leads to a pronounced basal narcosis throughout the subsequent anæsthesia. This prolonged action defeats any attempt to establish a truly balanced anæsthesia and will be referred to again. A trial of methohexital was suggested by reports of its short duration of action, prompt recovery and safety. Brief but certain effectiveness is frequently required for work in the out-patient department, where the sequence of untoward events that can follow thiopentone induction is notorious. This is of particular importance, for out-patient and maternity work are frequently conducted by those with the least experience of anæsthetic administration.

Most dental extractions under general anæsthesia in the chair are still carried out with nitrous oxide as the sole anæsthetic agent. Its use by the usual 'quick gas method' is not entirely satisfactory, as hypoxia is inevitable under these conditions. Anæsthesia can be achieved with-

out hypoxia by using nitrous oxide – oxygen mixtures, but the time taken is often inconveniently long. Mushin[1] stated that with 20 per cent oxygen and 80 per cent nitrous oxide induction will take from seven to ten minutes. If 15 per cent oxygen and 85 per cent nitrous oxide are used, satisfactory anæsthesia can usually be achieved without evidence of marked hypoxia, in from three to five minutes. This method is not always well tolerated by the nervous patient and may be associated with a period of excitement.

Bourne has successfully solved this problem of rapidly inducing dental anæsthesia without hypoxia by using cyclopropane delivered in a non-explosive mixture from a specially designed piece of apparatus. This excellent technique has two disadvantages; it is a 'one shot' method, and it has a high incidence of nausea and vomiting. Bourne[2] states that in one series 21 per cent of patients vomited and a further 12 per cent felt nauseated.

Our early experience with methohexital led us to believe that it could be used successfully as an induction agent for anæsthesia. We hoped that its very short action would eliminate the disadvantages previously experienced with other barbiturates.

Trials with the drug have been carried out in the St George's Hospital Group, at the Wembley Hospital and in the Dental Out-patient Department of the Royal Free Hospital.

CHEMISTRY

Methohexital sodium is α-dl-l-methyl-5-allyl-5 (1-methyl-2-pentynyl) barbiturate: It has no sulphur in its molecule and has two positions of unsaturation (acetylenic and olefinic).

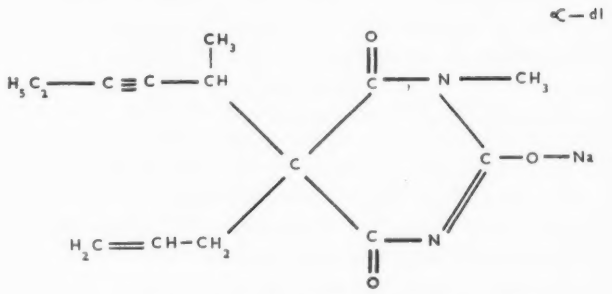

FIG. 1 Structural formula of methohexital sodium

The molecule contains two asymmetric carbon atoms so that two dl pairs exist capable of resolution into optically active d and l forms.

Gibson *et al*[3] studied the stereoisomers of this barbiturate and found

significant qualitative and quantitative differences. The most desirable configuration was the α-dl form called methohexital. This compound is stable in aqueous solutions at room temperature (25°C) for at least six weeks.

The pH of the 1 per cent solution is approximately 11.0.

Solutions of methohexital (sodium salt) should not be mixed with acid solutions *e.g.* atropine sulphate, dimethyl tubocurarine iodide and suxamethonium owing to the danger of precipitation of the barbiturate in the form of the sparingly-soluble free acid.

Plasma levels of methohexital may be determined by ultra-violet spectrophotometry[4 5].

PHARMACOLOGY

Greater potency than thiopentone (×2 to ×5). Graded single doses of methohexital given to mice, rats, dogs, cats and monkeys[4] have shown that methohexital is approximately twice as potent as thiopentone sodium. The action of the drug is characterised by smooth, rapid induction and by rapid recovery. The therapeutic index for rats and mice was also determined and was found to compare favourably with those for the other barbiturate anæsthetic agents. EEG studies[5] in cats put to sleep with methohexital or thiopentone were virtually indistinguishable.

Cumulation. Ten dogs were given 10mg per kg daily for twenty-eight days and then were sacrificed. Significant macro- or microscopic pathology was discovered neither in any of the organs nor in the bone marrow. The method of Wyngaarden *et al*[6] was used to evaluate the cumulative effects. Although methohexital does exhibit some cumulation, this is considerably less marked than with the thiobarbiturates.

Shorter duration of effect (half as long). It has been demonstrated[3] that for prolonged anæsthesia the greater duration of effect of the thiobarbiturates has less influence (on the total dose) than the greater potency of methohexital. Since the total dose is dependent upon both duration and potency, prolonged anæsthesia would require larger doses of methohexital than of the thiobarbiturates despite the greater potency of the former. This is borne out in practice. However, recovery following the thiobarbiturates used in this way is prolonged.

Prompt recovery. Under identical conditions[3] using EEG to control the depth of anæsthesia, plasma concentration of the methohexital to produce a given level of anæsthesia was approximately one-half that required by the thiobarbiturates (*i.e.* the methohexital is twice as potent). During recovery the concentration of the methohexital in the blood decreased rapidly and none could be identified after twenty-four hours; whereas at that time the level for the thiobarbiturates was

approximately 35 per cent of the level present at the end of the third hour of anæsthesia. (That is to say methohexital was more rapidly metabolised than was the thiobarbiturate). Other studies in dogs[7], indicate that methohexital is approximately two-and-a-half times as potent as thiopentone. (We have carried out similar studies in man, using methohexital in sleep doses to disappearance of eyelash reflex, on seventy-five cases and found the ratio of methohexital to thiopentone to be 2.8:1).

In prolonged anæsthesia in dogs, using a standard dose of 10mg per kg, there was no significant change in either blood pressure, or respiration or electro-cardiogram. No impairment of hepatic or renal function could be demonstrated by the para-aminohippuric acid and creatinine clearance tests.

Metabolic studies in dogs for recognition of the major metabolite of methohexital in the urine (Deininger method[8]) showed that it is comparable to those reported by Maynert[9] for pentobarbitone and amylobarbitone[10]. Brodie *et al*[5] found that the tissue distribution differs from that of thiopentone, in that the fat/plasma concentration ratio is much less than with thiopentone.

CLINICAL STUDIES

Clinical studies have been undertaken to compare methohexital with other intravenous anæsthetics[11 12 13 14 15 16]. Redish *et al*[12] showed that 134mg of 1 per cent methohexital were equivalent to 410mg of 2.5 per cent thiopentone. That is to say that methohexital is three times as potent as thiopentone and recovery from the former drug was significantly more rapid. Twitching and other muscular movements were noted in two patients receiving methohexital and both drugs were associated with grade one EEG dysrhythmia[12].

EEG studies on twelve patients before, during and after anæsthesia with intravenous methohexital[13] showed that its effects on the EEG recordings were minimal. Wyant *et al*[16] noticed a relatively long post-induction apnœa with the 1 per cent solution. This has not been reported elsewhere and was not found in our series.

DOSAGE

In one study[14] the amount of methohexital required for induction of anæsthesia had an average of 70mg ranging, in a series of 285 patients, from 50–220mg in 1 per cent solution. Of the total of over 10,000 administrations reported here – 100mg of the 1 per cent solution of methohexital were used for the purpose of inducing anæsthesia in the first thousand adult patients, their weights ranging from 6–18 stones. In every case induction was accomplished satisfactorily, even at the

Table 1

Distribution of cases

General and orthopædic out-patients		1200
Abdominal	major	1000
	minor	2000
Orthopædic	major	103
	minor	133
Emergencies (abdominal and orthopædic)		111
ENT	major	224
	minor (mostly children)	600
Plastic	adults	13
	children	7
Neurosurgical		11
Dental in-patients		23
Dental out-patients		373
Ophthalmic	adults	85
	children	109
ECT		4000
Thoracic	adults	37
	children	4
Cardiac	adults	9
	children	2
Vascular grafts and disobliterations		19
Maternity	forceps	53
	cæsarian sections	15
Bronchograms in children		11
Unclassified – including: thyroids, mastectomies, amputations, mesenteric thrombosis (1), varicose veins, branchial cysts (2) and laryngectomies (3)		99
	TOTAL	10,142

extremes of both weight and age. The considerable overdosage at these extremes demonstrated that there was a very wide margin of safety and also that a dose of 100mg need not be exceeded. Subsequent cases, including women in labour and children, were given doses sufficient to induce sleep. These doses varied from 20mg for a child of 4 years weighing three stones or a patient of 87 weighing six stones, to 100mg in a man of 33 weighing eighteen stones. The average dose was approximately 70mg and this compares favourably with other reports[14].

The patients drifted quietly into sleep (neither as rapidly nor as abruptly as with thiopentone) with little or no disturbance of respiratory rhythm. Apnœa following injection of methohexital has been reported. Taylor and Stoelting[17] record apnœas of up to three minutes duration in 17 per cent of cases from a total of 3,340 administrations and relate the duration of apnœa to the effectiveness of premedication and the rate of injection of methohexital. In our series there was no instance of apnœa; and, indeed, when 200–500mg were given repeatedly (by one of us) to goats and six-week old calves for induction of anæsthesia and intubation, there was still a continuance of regular respiration.

The period of sleep was short, lasting some two or three minutes in

the premedicated patient and somewhat less in the unpremedicated and sometimes apprehensive out-patient. The average induction dose for the majority of patients (*i.e.* those within the 8–12 stone weight range) was 70mg±15mg; approximately 7mg per stone of body weight.

On no occasion did the induction dose exceed 100mg and this would appear therefore to be a generous upper limit applicable to all types of cases.

Occasionally some fibrillary muscle twitching is noticed, particularly in the masseters. These muscular movements, finer than those evoked by thiopentone, are of short duration and appear to be of no significance. Extravenous injection produces slight erythema of only a few hours duration; it is not associated with pain and requires no treatment. Intra-arterial injection in calves evoked no undesirable response. Some 50 per cent of unpremedicated patients are aware of some sensation along the course of the injected vein[17]. In a few instances this awareness would seem to indicate pain, but as this sensation is not recollected in the post-operative period nor associated with phlebitis, it is of no significance.

MANAGEMENT

To achieve optimum results methohexital requires to be injected continuously and fairly rapidly. Moreover, when using a drug of such short duration, no time must be wasted in arranging an adequate maintenance routine. The smooth transition, without apnœa, from wakefulness to sleep enables inhalation mixtures to be taken from the moment of induction and they should be ready for immediate use.

Methohexital does not appear to increase vagal tone; bronchospasm has not been encountered and it would seem that laryngeal sensitivity is not enhanced. When this induction agent is followed by a short-acting relaxant (*e.g.* for intubation), adequate steps must be taken to ensure the continuance of sleep, for by the time the relaxant effect is exhausted, the patient may have recovered wakefulness.

Methohexital has but minimal effect on the blood pressure even when the dose is in excess of the minimal requirements. It can, therefore, be used with safety for inducing anæsthesia in cardiac cases. However, if subsequent doses are administered during the course of established anæsthesia a fall in blood pressure results; but this is neither so prolonged nor profound as that induced by thiopentone.

GENERAL SCHEME

A precise and rapidly cleared induction agent is of the utmost value in attempting a balanced anæsthesia. After induction with methohexital,

subsequent intubation and with control of relaxation dictated by the surgical requirements, of fluid balance and of pulmonary carbon-dioxide tension, then any variation in pulse-rate and blood pressure can be directly referred to analgesic requirements alone.

For maternity cases the scheme is appropriately modified, cyclopropane being used to provide analgesia, intubation performed as routine and respiration assisted up to the time of the birth.

The scheme is also modified to suit the out-patient for electroconvulsive therapy. Induction is followed by a short-acting relaxant; as soon as the effect of the latter is obvious a shock is administered, and the patient's lungs are inflated with oxygen should this be necessary.

PREMEDICATION WITH METHOHEXITAL

Rectal premedication in children can be effectively and safely accomplished with methohexital using 6–8mg per lb of body weight. Sleep of ten to fifteen minutes duration follows some ten to fifteen minutes after administration. Using the 1 per cent solution there is no irritation of the rectum or colon; but in addition to the usual objections to this method there is the undue bulk of the fluid – 33ml for instance are required for a child of three stones weight.

Oral premedication was tried in children, using either methohexital solution or tablets. The solution was found to be unsatisfactory because of the volume of fluid required, the horrid taste which could not be successfully disguised and the relatively large dose required for a very short period of even light sleep. The tablets (50mg) were equally unsuccessful owing to their bulk (*i.e.* the number required) and their unpleasant taste, the former being by far the more important factor.

DENTAL SCHEME

Our main objects were to obtain quiet dental anæsthesia without hypoxia and to achieve a rapid recovery.

Following the usual pre-operative precautions and examination of the mouth, a prop was inserted and methohexital given intravenously as rapidly as possible. The dose, 5mg per stone body weight, was the minimum which experience had shown consistently to produce sleep for two minutes. To conform with conditions in the average dental surgery the patients were not weighed, but their weight was assessed on their appearance and on information given. As soon as the patient was asleep a mixture containing 15 per cent oxygen and 85 per cent nitrous oxide was administered with a nasal mask. The time taken for anæsthesia to ensue was noticeably longer than that experienced with equipotent doses of thiopentone. The gas and oxygen was delivered

from a Walton machine, with high pressure and the bag turned off, so as to eliminate rebreathing. One-and-a-half to three minutes were required in the majority of cases to establish satisfactory conditions for operation. At this stage the oxygen percentage was increased to 20 per cent.

DISCUSSION

Methohexital is a drug with many virtues. Its chemistry and pharmacology have been fully explored ; the clinical trials were started tentatively but have developed as the potentials of the drug become understood. The dose of methohexital is closely linked with the strength of the preparation used, the function expected from the drug and whether or not the patient has received some form of premedication. In the dental trials reported here (R A G) the intravenous sleep dose for the unpremedicated out-patient was found to be 5mg per stone of body weight, while in the premedicated in-patient (D J C) it was established at 7mg per stone of body weight. The discrepancy can be accounted for in part by the absence of exact weight records for the out-patient group of dental cases and in part by the initial one thousand general surgical cases that received 100mg irrespective of their weight. These figures are approximately one-half of those required for a 'sleep dose' of thiopentone, (*i.e.* the methohexital is twice as potent as the thiopentone) so that these clinical findings do tally with the earlier work[3].

The complete absence of apnœa is a particular feature of methohexital induction using the dosage we have described. In this our findings are at variance with those of Wyant *et al.*[15] who, in their only adverse comment of the compound, refer to the 'prolonged apnœa in whatever concentration it is used'. We have not encountered a single instance of this.

Twenty of the sixty-eight maternity cases received 100mg each and are included in the initial one thousand cases. The remainder received 7mg per stone body weight. In this admittedly small series there was no instance of variation of the fœtal pulse rate nor of subsequent depression of infant respiration. Trials are proceeding in order more accurately to define the clinical impression that methohexital is superior to thiopentone in these respects. The absence of any marked fall in blood pressure following methohexital induction for obstetric surgery is of some importance.

Recovery from ECT following methohexital compares very favourably with that following thiopentone. The latter patients awake somewhat disorientated, confused and are not infrequently intermittently sleepy and inco-ordinate for a period of hours. Following methohexital induction for ECT the patient wakes in one to ten minutes quietly and fully, so that he tends to continue with whatever was occu-

pying him immediately previous to the ECT and within fifteen minutes is fit to leave. Again Taylor and Stoelting[17] reporting on the post-operative condition of their series state that many were orientated and mentally clear. There is no doubt in the minds of those, particularly the nursing staff, who have conducted ECT with both drugs that methohexital is preferable. There is no doubt also that recovery time from the short-acting relaxants is reduced following methohexital as compared with thiopentone. Moreover, in the case of methohexital the recovery time appears to be unrelated to the dose, whereas it is closely correlated with it in the case of thiopentone. This may be accounted for by the apnœic effect of a larger dose of thiopentone, and its anti-cholinesterase effect, together with any tendency to over-

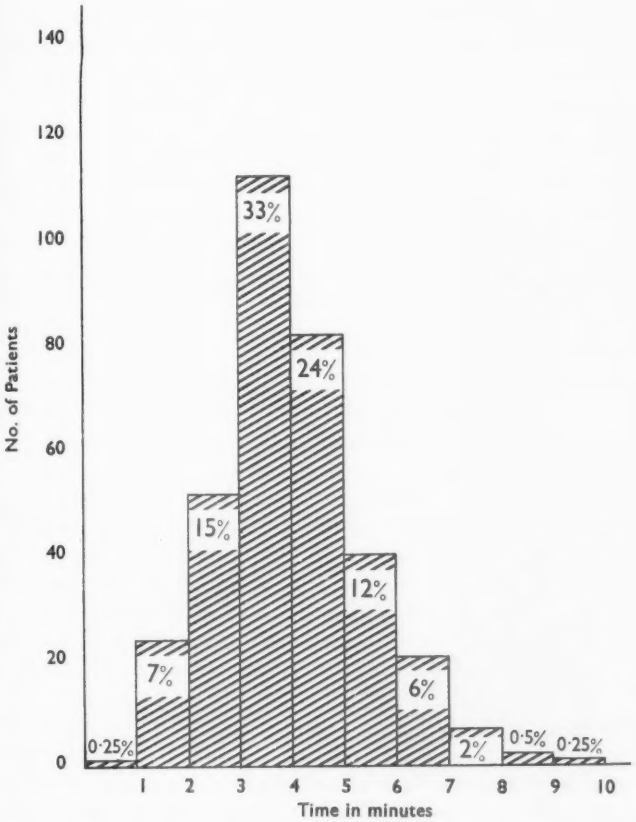

FIG. 2 Recovery time from receiving injection until leaving dental chair

inflation which by inducing a temporary respiratory alkalosis retains the thiopentone effect.

Of the 373 dental patients reported, 215 were adult females, 121 adult males, and 37 children under fourteen (the youngest aged six-and-a-half years). In only twelve cases was it necessary to supplement the nitrous oxide with any other inhalational agent. In seven of these, nasal breathing was not easy to establish and trichlorethylene was used. In one case ethyl chloride on a mouth pack was the only way to maintain anæsthesia in a persistent mouth-breather. A further four patients required trichlorethylene to establish smooth anæsthesia in spite of satisfactory nasal respiration.

A restraining strap was not used on any of the patients, although many were tough labourers. In only thirty-four of the patients was any degree of manual restraint required.

Two periods during recovery were recorded : the time from receiving the injection until the patient was capable of leaving the chair for the

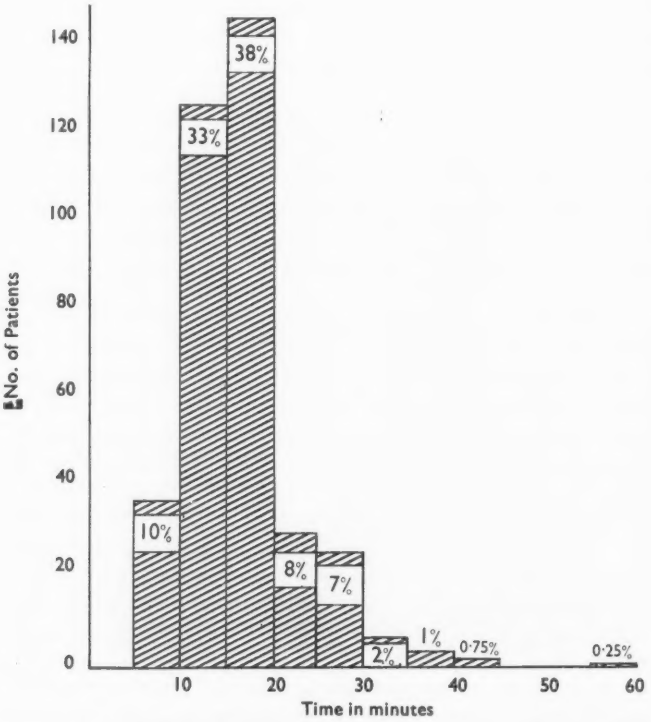

FIG. 3 **Recovery time from receiving injection until leaving clinic**

recovery room with the assistance of one nurse (FIG. 2) and the time from receiving the injection until it was considered that recovery was complete and that it was safe for the patient to go home unaccompanied (FIG. 3). Ninety-six per cent of the patients were deemed fit to return home within half-an-hour, 84 per cent within twenty minutes. Only one patient caused us any inconvenience; but then behaved in a similar fashion following a subsequent extraction under nitrous oxide alone. Ninety-two per cent of the patients were able to leave the dental chair within six minutes of receiving the injection.

Reactions to this type of anæsthesia were solicited from one hundred consecutive patients by means of a questionnaire, aimed at discovering personal responses and any complications of recovery which might have occurred after leaving the clinic. All these forms were completed and were returned the next day.

Only 7 per cent of the patients who had previously had nitrous oxide inductions for dental anæsthesia preferred that method, the majority of the remainder expressed their preference for the methohexital.

Sixteen patients felt the need to lie down on returning home, thirteen of these were female, one was male and two were children. All except one of these patients however expressed their preference for this method. The rest of the patients were able to return immediately to their usual occupation. Two patients complained of feeling extractions, although they made no complaint at the time. Ninety-four per cent of the patients said that they felt quite normal when they left the clinic. Of the 6 per cent who did not feel normal on the way home, three complained of feeling nauseated, one felt tired, one dizzy and one faint. None had complained when given permission to go.

Blood pressures were taken on a large number of patients during the procedure and no profound falls were noticed.

Five per cent of the patients vomited during recovery, and 14 per cent felt nauseated. None of the general surgical patients suffered postoperative nausea to a degree that made them complain. This low incidence was probably related to the premedication, which contained promethazine 1 mg per stone in every case and to the use of lethidrone[18] reducing any morphine-like effect. Of the dental and other miscellaneous out-patient cases approximately 5 per cent suffered some postanæsthetic nauseous interlude while from among all the ECT cases only one was repeatedly sick (he continued to be so when the induction was achieved with thiopentone). The incidence of vomiting following the use of methohexital for dental extractions was encouragingly low. We feel that 5 per cent is probably the irreducible minimum that can be obtained in unpremedicated patients, by any method.

Spontaneous coughing (1 per cent), hiccough and laryngospasm (<1 per cent, usually mild and relieved by a further injection of methohexi-

tal and removal of any exciting stimulus) following induction with methohexital have been reported by Taylor and Stoelting[17]. We have not met with any of these complications using the methods outlined above. It is essential that methohexital be used solely to induce sleep (not for narcosis) and that analgesia with another agent be established within the period allowed by the sleep dose. The above-mentioned complications are thus met with during the period of awakening or lightening from the sleep dose and particularly in the presence of an exciting stimulus. A scheme of balanced anæsthesia is required so that the effect of one agent does not subside before another has taken over all or part of its function.

SUMMARY

Recovery following methohexital is a notable feature, being rapid, complete and notably free from side-effects[15]. These features have been discussed in relation to the value of the drug as an induction agent, for obstetric surgery, and its place in helping to establish a balanced anæsthetic technique.

Methohexital has great promise for use in out-patients provided it is used solely as an induction agent and in the small doses described. The rate of recovery of the patients seemed to be quicker than from any barbiturate previously investigated.

Although the time taken to prepare the patient for dental extraction was longer than with pure nitrous oxide, the smoothness of the procedure and the absence of hypoxia more than compensated for this. Out-patients seem to welcome this type of anæsthesia.

Further studies are aimed at assessing co-ordination following recovery from the drug and will be reported later.

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References

[1] MUSHIN, W. W. (1952). *Brit. Med. J.*, *1*, 431. Anæsthesia for Minor Procedures.
[2] BOURNE, J. G. (1952). *Lancet*, *2*, 705. Cyclopropane Anæsthesia for Dental Extractions.

[3]GIBSON, W. R., DORAN, W. J. and WOOD, W. C. To be published.

[4]BRODIE, B. B., BERNSTEIN, E. and MARK, L. C. (1952). Role of Body Fat in Limiting Duration of Action of Thiopental, *J. Pharmacol. & Exper. Therap.*, *105*, 421.

[5]BRODIE, B. B., BURNS, J. J., MARK, L. C., LIEF, P. A. and PAPPER, E. M. (1953). Fate of Pentobarbital in Man and Dog and Method for Its Estimation in Biologic Material, *J. Pharmacol. & Exper. Therap.*, *109*, 26.

[6]WYNGAARDEN, J. G., WOODS, L. A., RIDLEY, R. and SEEVERS, M. H. (1949). Anesthetic Properties of Sodium 5-Allyl-5-(1-Methyl-Butyl)-2-Thiobarbiturates (Surital) and Certain Other Thiobarbiturates in Dogs. *J. Pharmacol. & Exper. Therap.*, *95*, 322.

[7]BELLVILLE, J. W. Personal communication.

[8]DEININGER, R. (1955). Papierchromatographischer Nachiveis der Barbitursauren, Arzneimittel-Forsch. *5*, 472.

[9]MAYNERT, E. W. and DAWSON, J. M. (1952). Ethyl-(3-Hydroxy-1-Methyl butyl) Barbituric Acids as Metabolites of Pentobarbital. *J. Biol. Chem.*, *195*, 389.

[10]MAYNERT, E. W. (1952). Ethyl-(3-Hydroxyisoamyl) Barbituric Acid as the Principal Metabolite of Amytal. *J. Biol. Chem.*, *195*, 397.

[11]HEATON, C. E. (1956). Clinical Experiences with 1-Methyl-5-Allyl-5-(1-Methyl-2-Pentynyl) Barbituric Acid Sodium Salt, Current Res. in Anesth. and Analg., *35*, 522.

[12]REDISH, C. H., VORE, R. E., CHERNISH, S. M. and GRUBER, C. M. JR. (1958). A Comparison of Thiopental Sodium, Methitural Sodium, and Compound 25398 in Oral Surgery Patients. *Oral Surg., June.*

[13]RIFFIN, I. M., PREISIG, R. and SCHWARTZ, B. Clinical Evaluation of a New Ultra-Short Acting Barbiturate Compound 25398. Bull. Mountainside Hosp. Montclair, N.J. Publication pending.

[14]STOELTING, V. K. (1957). The Use of a New Intravenous Oxygen Barbiturate 25398 for Intravenous Anesthesia (A Preliminary Report). *Anesth. & Analg.*, *36*, 49.

[15]WYANT, G. M., DOBKIN, A. B. and AASHEIM, G. M. (1957). Comparison of Seven Intravenous Anæsthetic Agents in Man. *Brit. J. Anæsth.*, *29*, 194.

[16]DOBKIN, A. B. and WYANT, G. M. (1957). The Physiological Effects of Intravenous Anæsthesia in Man. *Canad. Anæsth. Soc. J.*, *4*, 295.

[17]TAYLOR, C. and STOELTING, V. K. (1960). *Anesthesiology*, Jan–Feb, *21*, 1, pp.29–34.

[18]COLEMAN, D. J., HARGROVE, R. L. and JONES, P. O. (1958). *Anæsthesia*, *13*, 1, January.

Blood loss in pædiatric surgery

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Experience has proved that clinical assessment of blood loss during surgery is a fallacious guide to blood replacement therapy[4 10 13]. Such clinical assessments invariably underestimate the volume of blood lost. These errors of judgment may be as large as 50 per cent[7] and 200 per cent[2]. In pædiatric surgery in general and particularly in the neonatal group such errors will jeopardize the efficacy of surgery and the lives of the young patients. A method of blood loss assessment, however simple, which aims at reducing the factor of personal judgment with its wide variations to a minimum, must be regarded as an essential component of sound pædiatric surgical practice.

Blood loss in adult surgery has been extensively reported on since Gatch and Little[4] and Wangensteen[9] pioneered investigations based on the colorimetric and gravimetric techniques respectively. During the period 1924 to date, upwards of 3000 cases from all branches of adult surgery have been reported on in the literature. These results are highly instructive and anæsthetists should be familiar with the mean expected blood loss in the various surgical procedures. It is accepted that the anæsthetist in his moment to moment control of the patient's physiological equilibrium is in the best position to assess the degree of blood loss. At their best such clinical assessments are fallible even after considerable clinical experience. Anæsthetists in training will be well advised to study the invaluable reports of past investigators into blood loss during surgery and to use them as a guide in daily clinical practice.

Whilst there exists a safety margin of 500ml or more in the fit adult, the loss of 20ml in an infant weighing six pounds may make considerable difference to the ultimate result of surgical intervention. It is imperative that the variable factor of personal judgment should be eliminated as far as possible in pædiatric surgery.

It is surprising to find only one report[8] in the literature on the subject of blood loss in pædiatric surgery. In this report Rickham discusses the pitfalls of the gravimetric technique and the undoubted superiority of the colorimetric method as an accurate estimate of the surgical blood loss. The latter however is a time consuming procedure which offers practical difficulties should one wish to incorporate it into daily operating theatre routine. In addition it lacks the advantage of guidance during an operation in that the final assessment can only be made immediately post-operatively. A simple gravimetric technique, despite its shortcomings, has the advantage of offering guidance during the operative procedure and this is superior to the practice of guesswork, and does so without additional strain on already limited theatre staff.

MATERIAL PRESENTED

The present study covers 443 operations on neonates, infants and children drawn from European, Cape Coloured and African population groups. The majority of patients belonged to the latter two population groups. This fact is stressed because those children invariably present with a poor nutritional background and have an extremely limited reserve, if any at all, to resist the trauma of illness. In a survey of pre-school going children Lanzkowsky *et al*[5], demonstrated the extent to which iron deficiency anæmia exists in Cape Town. The incidence of anæmia was 64.9 per cent for Cape Coloured children and 66.7 per cent for African children whilst observations on a small number of white children from a high socio-economic group showed hæmoglobin levels almost identical to those of Wintrobe's normal standard. The additional stress of surgical blood loss therefore immediately assumes far greater importance than it does in the average well nourished white child who comes from a sound socio-economic environment.

APPARATUS AND TECHNIQUE

A simple Avery weighing scale accurate to 5g balancing soiled swabs against standard control swabs. To avoid errors in operative 'swab counts' the controls were lightly stained with a distinctive colour and kept in the weighing cabinet.

Small soiled drapes were likewise balanced against unsoiled controls.

Direct measurement of the suction bottle component of blood loss.

Clinical estimate of blood spilled on to the larger drapes and surgical gowns.

The dry swab technique used throughout the series.

Expected normal blood volume of the patients calculated on the basis of 40ml per pound of body weight.

One ml of blood taken as weighing 1g for practical purposes.

RESULTS AND DISCUSSION

The results are tabulated to indicate : (A) The number of cases in each type of operation. (B) Blood loss expressed as a percentage of expected blood volume (EBV). (C) The range of blood loss in each operation; expressed as a percentage of EBV. (D) The number of cases transfused.

AGE AND WEIGHT DISTRIBUTION

Age and Weight distribution are summarised in FIGS. 1 and 2.

One hundred and seventy-six (39 per cent) cases received blood transfusions. In all instances replacement was made at the time of loss during surgery. A syringe and three-way tap technique was used for transfusion in neonates and infants and the standard drip infusion technique in the bigger children.

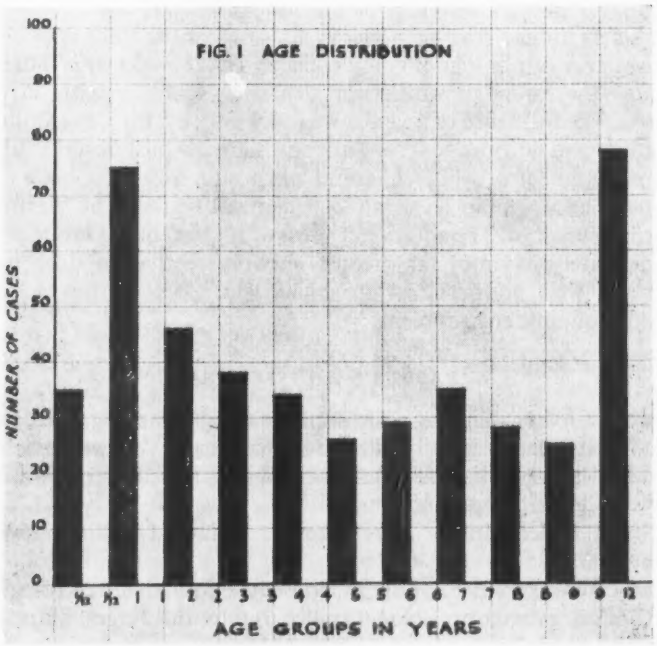


FIG. 1 Blood Loss in Pædiatric Surgery

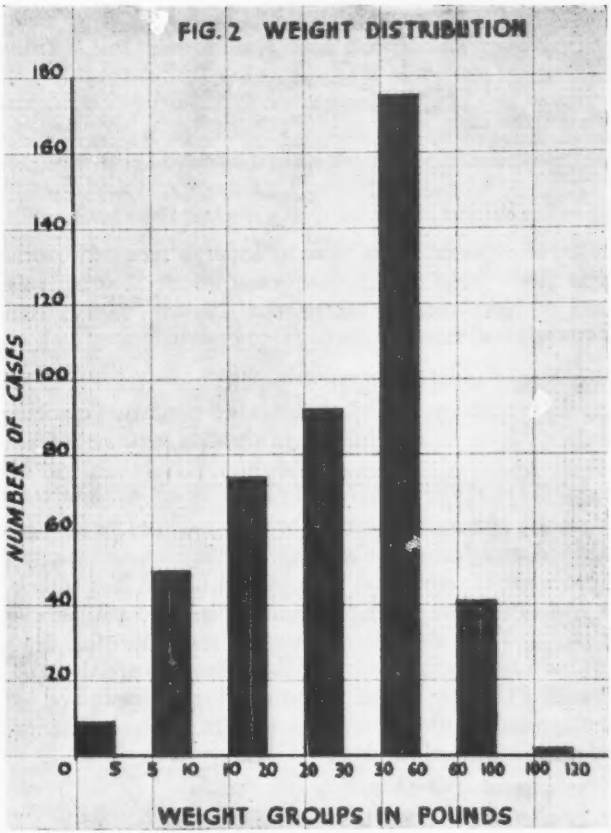


FIG. 2 Blood Loss in Pædiatric Surgery

PLASTIC SURGERY

Thiersch graft to burns

In our experience this division of surgery presents the most dangerous pitfalls to the unwary anæsthetist. Blood loss is severe and due to the use of electrically driven dermatomes the loss is precipitous ; occurring within the first five or ten minutes of the operation. Unless a drip infusion is already in place and blood readily available at a moment's notice the anæsthetist will be confronted with a severely shocked child and considerable difficulty in setting up a transfusion. Cardiac arrest during this critical period is a distinct danger and occurred in one of the cases in the present series[6].

Table 1
Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
Plastic surgery				
Thiersch graft to burns*	79	7	0.6–69	*42*
Pedicles and Z plasty	15	15.1	1.0–37	8
„ +Thiersch grafts*	5	*27.3*	4.0–52	5
Cheilorrhaphy	26	2.9	0.6–7.0	—
Palatorrhaphy	28	4.4	0.1–11.5	—
Toilet burn granulations*	5	*26.0*	1.9–*48*	3

These patients frequently have to undergo repeated operations in order to cover large burn areas. Serial losses of apparently minor volumes of blood per operation must be scrupulously replaced if gross anæmia and retarded healing is to be avoided.

Surgical toilet of burn area granulations

The results emphasize the fact that this is a procedure associated with a large blood loss. Too often it is regarded as a minor trauma.

ACUTE OSTEOMYELITIS; INCISION AND DRAINAGE

The exposure of bone through highly inflamed tissues means replacement blood transfusion for virtually all these cases where extensive exposure without tourniquet is employed. Blood loss is consistently severe and often even more precipitous than in the Thiersch graft to burns group. These children are severely toxæmic, often *in extremis*, by the time they present to our surgical colleagues and demand precise pre-operative therapy. Casual anæsthetic management and ignorance of the magnitude of blood loss to be anticipated can decidedly destroy any existing chances of survival.

Table 2
Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
I/D acute osteitis				
Femur*	26	*13.0*	2.0–38	*21*
Tibia	8	10.5	3.5–31	*5*
Humerus	5	9.2	2.0–13	*4*
Herniæ				
Inguinal herniotomy	43	1.2	0.1–5.7	—
Strangulated I.H. – reduction	2	1.6	—	—
„ + bowel resection*	2	*14.5*	—	2
Umbilical herniorrhaphy	4	1.3	0.5–1.6	—
UDT	18	1.1	0.3–7.0	—
ENT				
T's and A's	24	4.7	0.8–*9.0*	—
T's	2	3.2	—	—
A's	2	2.2	0.5–4.0	—
Mastoidectomy*	2	3.0	2.0–4.0	—

ENT SURGERY

The results in this division have been included to stress the need for considerably more data. The upper limit of blood loss in tonsillectomy and adenoidectomy reached 9 per cent of EBV which is patently near the indication for transfusion. The two mastoidectomy results are completely deceptive because one knows from clinical experience that blood loss can be severe and not infrequently these children need blood transfusions. One ventures to suggest that more extensive investigation into blood loss in pædiatric ENT surgery will indicate the need for a considerably higher incidence of replacement transfusions than has been our practice to date.

ABDOMINAL SURGERY

It is fortunate that one always anticipates high blood losses in abdominal surgery, thus being adequately prepared for transfusion therapy. The results indicate that most procedures entail blood transfusion. Particular attention is drawn to the 'apparently innocent' operations of ileostomy and closure of colostomy. The high blood loss in operations necessitating bowel resection is clearly illustrated by comparison between simple reduction of intussusception and volvulus

Table 3

Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
Abdominal				
Retroperitoneal tumour + bowel resection	1	*140.3*	—	1
Mesenteric tumour	2	14.9	12.8–17	2
Ovarian tumour (Granulosa Cell)	3	7.2	3.8–9.3	2
Splenectomy	2	5.0	3.6–6.4	1
Gastro-jejunostomy	1	16.0	—	1
Ileostomy	2	*20.0*	17.0–23.0	2
Colostomy	3	2.5	0.9–3.5	—
Closure of colostomy*	2	*21.5*	10.1–*33.0*	2
T/V colostomy + anal back cut	1	23.0	—	1

Table 4

Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
Abdominal				
Pyloromyotomy	12	2.4	0.7–3.1	—
Cardiomyotomy	2	*13.5*	12.0–15.0	2
Intussusception – reduction	8	4.1	1.0–7.0	2
„ – bowel resection*	4	*17.3*	2.0–22.0	4
Appendectomy	20	1.0	0.2–3.2	—
Recto-sigmoidectomy*	15	*26.9*	5.0–*53.0*	15
Obstruction				
Duodenal	8	20.5	11.0–37.0	8
Ileal	3	23.3	13.3–35.3	3
Volvulus – reduction	3	5.4	3.3–9	—
„ – bowel resection	1	*42.5*	—	1
Adhesions	6	*13.0*	2.3–18.0	5

and those procedures involving bowel resection. A similar difference is noticed in the case of strangulated inguinal hernia (TABLE 2).

Blood loss during recto-sigmoidectomy is consistently very high as indicated by the mean of 26.9 per cent. The danger here is increased by the background of a severely ill infant with marked electrolyte disturbance.

THORACO-ABDOMINAL PROCEDURES

Although blood loss during the repair of tracheo-œsophageal fistula can be classed as moderate the need for precise replacement cannot be over emphasized. Errors in blood replacement *per se* may invalidate

Table 5

Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
Thoraco-abdominal				
Tracheo-œsophageal fistula*	5	*14.2*	4.0–22.0	5
Hiatus hernia*	2	16.3	4.6–*28.0*	2
Traumatic œsophageal stenosis :				
By-pass – stage I	2	33.5	24.7–42.4	2
By-pass – stage II	2	7.3	5.9–8.8	2
Patent ductus arteriosus ligation	4	14.0	6.0–16.0	4
Pericardectomy	1	13.4	—	1

this highly skilled surgical procedure. We agree with Rickham[8] that blood replacement should finally be in excess of the assessed loss but not to the volume of 35–50ml as he suggests. We supplement the estimated loss by 10 per cent of the neonate's expected blood volume.

UROLOGY

Upwards of 95 per cent of major urological operations will necessitate replacement blood transfusion. The repair of ectopia vesicæ is

Table 6

Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
*Urological**				
Nephrectomy	6	14.3	5.2–*38.0*	4
Wilms' tumour	2	33.9	18.0–*49.0*	2
Repair ectopia vesicæ	6	*59.3*	7.0–*100*	5
Bladder resection	1	28.0	—	1
Re-implantation ureters	1	28.0	—	1
Pyeloplasty	1	10.3	—	1
Exploration kidney	1	12.2	—	1
Nephrostomy	1	9.5	—	1
Explore ruptured urethra	1	3.0	—	1

consistently the highest blood loss procedure in our series. The results in this division confirm those of Rickham[8] who pointed out that pædiatric genito-urinary surgery must be classed in the high blood loss group of procedures.

Table 7

Blood loss in pædiatric surgery

OPERATION	NO OF CASES	% MEAN LOSS OF EBV	% RANGE	NO BTF
Head and neck				
Cystic hygroma*	3	15.3	1.1–22.0	1
Branchial fistula	1	1.0	—	—
Thyroglossal cyst	1	2.0	—	—
Malignant tumour mandible	1	*31.0*	—	1
Retro-ocular hydatid	1	7.0	—	1
Enucleation eyeball	1	5.6	—	1
Dorsum-trunk				
Meningocele – spina bifida*	2	15.3	7.6–23.0	2
Excision lipoma*	2	6.6	2.0–*11.3*	1

SUMMARY

A study of gravimetric blood loss estimations is presented in a series of 443 pædiatric surgical operations.

It is established that the operations for Thiersch graft to burns, incision and drainage of acute osteomyelitis and major urological surgery involve considerable blood loss. The former two divisions present the greatest danger in that the precipitous nature of the loss is often not anticipated.

The importance of a poor nutritional state, relative to the stress of the presenting illness, anæsthesia and surgery is emphasized.

There exists a need for more extensive investigation into blood loss during pædiatric ENT surgery.

A simple practical gravimetric method of blood loss estimation during pædiatric surgery is presented. Although admittedly not ideal it has served to eliminate the fallacious guessing commonly termed 'clinical estimation of blood loss'.

Acknowledgements

I wish to thank Dr J.F.W. Mostert, Medical Superintendent, Red Cross War Memorial Children's Hospital, Rondebosch, Cape Town, for permission to present this report, Professor J.H. Louw, Head of the Pædiatric Surgical Unit and his colleagues for their kind co-operation and Dr A.B. Bull Jnr, Senior Consultant Anæsthetist to the Pædiatric unit, whose keen interest and invaluable advice directed this investigation.

References

[1]BARONOFSKY, IVAN D., TRELOAR, ALLAN E. and WANGENSTEEN, OWEN H. (1946). *Surgery*, *20*, 761.
[2]BONICA, JOHN J. and LYTER, CLINTON S. (1951). *Anesthesiology*, *12*, 90.
[3]CROOK, CLARENCE E., IOB, VIVIAN and COLLER, FREDERICK A. (1946). *Surg., Gynec. & Obst.*, *82*, 417.
[4]GATCH, W. D. and LITTLE, W. D. (1924). *J.A.M.A.*, *83*, 1075.
[5]LANZKOWSKY, P. and MCKENZIE, D. (1959). *S. Afr. Med. J.*, *33*, 21.
[6]MALHERBE, P. (1959). Personal communication.
[7]NADAL, JOSEPH W. (1939). *Univ. Hosp. Bull., Ann. Arbor.*, *5*, 74.
[8]RICKHAM, P. P. (1954). *Arch. Dis. Childhood*, *29*, 304.
[9]WANGENSTEEN, OWEN H. (1942). *Minnesota Med.*, *25*, 783.
[10]WINDFELD, P. (1936). *Acta Chir. Scandinav.*, *79*, 452.

NEW INVENTIONS

Unless expressly stated by the authors no claim to originality or priority is made and items given under this head include modifications to existing apparatus. The Editor regrets that space considerations make it impossible to publish correspondence on this subject

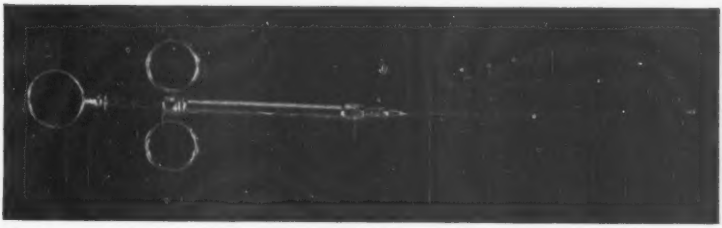
Local Analgesic Syringe

W. H. J. COLE

The photograph shows a syringe which I have used for eight years for spraying local analgesic solutions on the larynges and tracheas of unconscious patients. The apparatus consists of an all metal syringe of 1.6ml capacity fitted with thumb and finger grips, and a screw-on needle of 20 gauge size with a round ball of about $\frac{1}{8}$ inch diameter fitted to the free end to prevent possible tissue damage.

For the anæsthetist who has to carry his apparatus with him the syringe has the following advantages: it is light, compact, indestructible, may be sterilized by boiling or autoclaving, and, is readily refilled when necessary.

Photograph taken by the Victorian Eye and Ear Hospital Photographic Department (Australia).



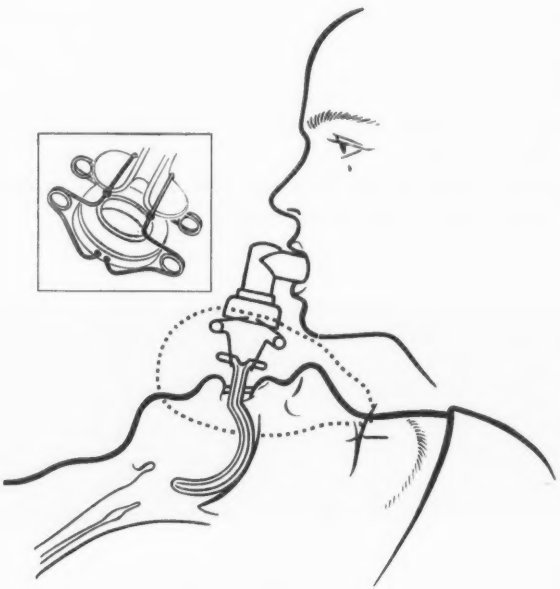
Airway Clip

B. G. B. LUCAS

When using any of the commonly available oxygen resuscitation equipment, it is sometimes helpful to insert a pharyngeal airway prior to placing a mask over the subject's face.

An airway, however, is not always available in an emergency because it is not an integral part of the apparatus and a rigid, permanent attachment of an airway to the inside of a facemask makes insertion of the former difficult and upsets the correct placing of the latter.

By connecting the two together by means of a stainless steel spring, 20SWG, these difficulties are overcome. The complete mask and airway, with an angle mount, can also be used for mouth-to-mouth resuscitation. I am grateful to Mr E. P. Childerhouse, who suggested this idea to me and has made the first prototype.



Endotracheal connections for pæ'iatric use

A modification of Nosworthy's connection for use in children

P. F. KNIGHT

Most angled connections for use with the smaller sizes of endotracheal tube in pædiatric anæsthesia, effectively prevent the passage of either an introducing stylet or a suction catheter. These requirements are met admirably in adult practice by connections of the Nosworthy type, and the modification shown in FIG. 1 is virtually a scaled down edition of this.

FIG. 1

The set shown was manufactured for the author by Medical and Industrial Equipment Ltd, 10–12 New Cavendish Street, London W1, and consists of a single angled proximal limb (flanged for firm attachment to a T-piece, as shown in FIG. 2) fitting by a taper joint any of four tube mounts, which are graded in size to carry Magill tubes from size 00 to 5.

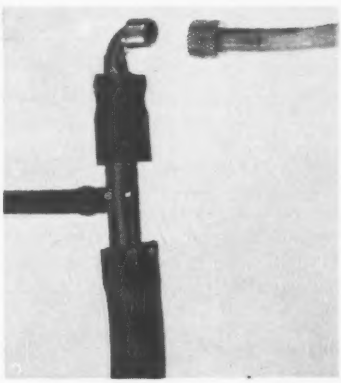


FIG. 2

The dead space volume of this connection is very small, and in addition to allowing easy use of the sucker it facilitates rapid attachment of endotracheal tube to anæsthetic gas supply.

I am indebted to the Photographic Department of St Mary's Hospital for the illustrations.

Anæsthetic literature

APPARATUS

Intermittent positive pressure respirator. FRUMIN, M. J., LEE, A. S. J. and PAPPER, E. M. (1960) *Anesthesiology*, *21*, 220 (March).

The polymask as a means of administering oxygen. CATTERALL, M. and SNOW, M. (1960) *Brit. med. J.*, *1*, 1254 (April 23).

A transistorised drip indicator. DANBURY, R. (1960) *Lancet*, *1*, 905 (April 23).

Instrumentation and monitoring during open-heart surgery. STEWART, E., OSBORN, J. J. and GERBODE, F. (1960) *Arch. Surg.*, *80*, 677 (April). 7 refs.

An easy method for the determination of the pCO_2 of the blood. MENDEL, D. (1960) *Lancet*, *1*, 807 (April 9).

New, pulse-giving, hydraulic pump for extracorporeal circulation. SØRENSEN, A. H. (1960) *Lancet*, *1*, 959 (April 30).

Disinfection of anæsthetic face masks. MACCALLUM, F. O. and NOBLE, W. C. (1960) *Brit. J. Anæsth.*, *32*, 192 (April). No refs. (Water at 60–70° C is best.)

Evolution of endotracheal and endobronchial intubation. WHITE, G. M. J. (1960) *Brit. J. Anæsth.*, *32*, 235. 32 refs.

ARTICLES OF GENERAL INTEREST

Liability of the anesthesiologist and the surgeon. WASMUTH, C. E. (1960) *J. Amer. med. Ass.*, *172*, 1473 (April 2).

Symposium on pædiatric anæsthesia. Various Authors (1960) *Brit. J. Anæsth.*, *32*, 95–140 (March).

Joseph Clover and the contributions of surgery to anæsthesia. LEE, J. A. (1960) *Ann. Roy. Coll. Surg.*, *26*, 280 (May). 61 refs.

COMPLICATIONS OF ANÆSTHESIA AND OPERATION

Post-operative nausea and vomiting IV: factors related to post-operative nausea and vomiting. BELLVILLE, J. W., BROSS, I. D. J. and HOWLAND, W. S. (1960) *Anesthesiology*, *21*, 186 (March). 8 refs.

Circulatory changes due to open pneumothorax in surgical patients. LI, T. H., RHEINLANDER, H. F. and ETSTEN, B. (1960) *Anesthesiology*, *21*, 171 (March). 25 refs.

The anesthetic significance of serotonin secreting carcinoid tumours. STONE, H. H. and DONNELLY, C. C. (1960) *Anesthesiology*, *21*, 203 (March). 57 refs.

Pre-operative use of anti-emetics. KEATS, A. S. (1960) *Anesthesiology*, *21*, 213 (March). Editorial.

Anæsthesia in dystrophia myotonica. KAUFMAN, L. (1960) *Proc. Roy. Soc. Med.*, *53*, 183 (March). A review of the hazards of anæsthesia. 37 refs.

Respiratory obstruction in hæmophilic patients. LEATHERDALE, R. A. L. (1960) *Brit. med. J.*, *1*, 1316 (April 30). 24 refs.

Vomiting by out-patients after nitrous oxide anæsthesia. BODMAN, R. I., MORTON, H. J. V. and THOMAS, E. T. (1960) *Brit. med. J.*, *1*, 1327 (April 30).

Continuing hazard of air embolism during pressure transfusions. RUESCH, MCL., MIYATAKE, S. and BALLINGER, C. M. (1960) *J. Amer. med. Ass.*, *172*, 1476 (April 2). 16 refs.

Long-term follow-up of patients who received 10,098 spinal anæsthetics. VANDAM, L. D. and DRIPPS, R. D. (1960) *J. Amer. med. Ass., 172*, 1483 (April 2). IV. Neurological disease incident to traumatic lumbar puncture during spinal anæsthesia. 14 refs.

Post-operative nausea and vomiting. BELLVILLE, J. W., HOWLAND, W. S. and BROSS, I. D. J. (1960) *J. Amer. med. Ass., 172*, 1488 (April 2). III. Evaluation of the anti-emetic drugs fluphenazine (prolixin) and promethazine (phenergan) and comparison with triflupromazine (nesprin) and cyclizine (marezine). 8 refs.

Bucking and bronchospasm as problems of anesthesia. SMITH, R. H. and VOLPITTO, P. P. (1960) *J. Amer. med. Ass., 172*, 1499 (April 2). 18 refs.

Cardiac arrest during anæsthesia and surgery. ENGELL, H. C., PRICE, J. and SECHER, O. (1959) *Acta Anæsth. Scand.*, Supplementum 1.

An analysis of deaths in operating room and within twenty-four hours of surgery. SCHAPIRA, M., KEPES, E. R. and HURWITT, E. S. (1960) *Curr. Res. Anesth., 39*, 149 (March–April). 4 refs.

Classification of operating room mortality. AMENT, R. (1960) *Curr. Res. Anesth., 39*, 158 (March–April). 13 refs.

Post-operative nausea and vomiting: the response to anti-emetic drugs with special reference to cyclizine and perphenazine. ROBERTS, H. (1960) *Canad. Anæsth. Soc. J., 7*, 116 (April). 9 refs.

Cardiac arrest following administration of a high concentration of halothane vapour. BAXTER, V. T. (1960) *Brit. J. Anæsth., 32*, 171 (April). 29 refs.

Atropine in laryngeal spasm. ROSEN, M. (1960) *Brit. J. Anæsth., 32*, 190.

Sore throat after anæsthesia. CONWAY, C. M., MILLER, J. S. and SUGDEN, F. L. H. (1960) *Brit. J. Anæsth., 32*, 219 (May). 2 refs.

GENERAL ANÆSTHETIC PROCEDURES

Re-discovery of air for anæsthesia in thoracic surgery. POPPELBAUM, H. F. (1960) *Proc. Roy. Soc. Med., 53*, 289 (April).

X-ray study of passage of air through the pharynx in anæsthetised patients. RUBEN, H., BENTZEN, N. and SAEV, S. K. (1960) *Lancet, 1*, 849 (April 16).

Discussion on the value of controlled respiration in neuro-surgery. HUNTER, A. R. and INGLIS, J. MCN. (1960) *Proc. Roy. Soc. Med., 53*, 365 (May). 29 refs.

Anæsthetic management of penetrating wounds of the heart. SCHAEFER, H. C. and DEVAULT, M. (1960) *J. Amer. med. Ass., 172*, 1913 (April 23). 15 refs.

Problems related to open heart operations in children (Part I). SMITH, R. M. and ENGINEER, E. H. (1960) *Curr. Res. Anesth., 39*, 104 (March–April). 9 refs.

Anæsthesia for open heart surgery. MENDELSOHN, D., MACDONALD, D. W., NOGUEIRA, C. and KAY, E. B. (1960) *Curr. Res. Anesth., 39*, 110 (March–April). 7 refs.

Clinical studies of new and old hydrocarbons. SIEBECKER, K. L., BAMFORTH, B. J., STEINHAUS, J. E. and ORTH, O. S. (1960) *Curr. Res. Anesth., 39*, 180 (March–April). 4 refs. (A comparison of chloroform and halothane.)

Anæsthesia for ligation of patent ductus arteriosus in children. CONN, A. W. and JUNKIN, C. I. (1960) *Canad. Anæsth. Soc. J., 7*, 169 (April). 18 refs.

Anæsthesia for cardiac catheterisation in children. ADAMS, A. K. and PARKHOUSE, J. (1960) *Brit. J. Anæsth., 32*, 69 (February). 27 refs.

The myasthenic state and the myotonic syndrome. MCCLELLAND, R. M. A. (1960) *Brit. J. Anæsth., 32*, 81 (February). 45 refs.

HYPOTHERMIA

Renal blood flow in moderate hypothermia. ONNIS, M., BOUNOUS, G. and SHUMACKER, H. B. (1960) *Arch. Surg.*, *80*, 593 (April). 14 refs.

Prevention of ventricular fibrillation during profound hypothermia by quinidine. JOHNSON, P. *et al* (1960) *Ann. Surg.*, *151*, 490 (April). 7 refs.

MUSCLE RELAXANTS

A method of monitoring muscular relaxation by the integrated abdominal electromyogram. FINK, B. R. (1960) *Anesthesiology*, *21*, 178 (March). 22 refs.

Dual neuromuscular block in man. CHURCHILL-DAVIDSON, H. C., CHRISTIE, T. H. and WISE, R. P. (1960) *Anesthesiology*, *21*, 144 (March). 10 refs.

OBSTETRICAL ANÆSTHESIA AND ANALGESIA

The anti-emetic action of fluothane: A comparative study in obstetrical anæsthesia. NOVOA, R. R. (1960) *Canad. Anæsth. Soc. J.*, *7*, 109 (April). 11 refs.

Mepazine (pacatal), meperidine (pethidine) and dihydrocodeine in labour. PARKIS, I. E. (1960) *Canad. Anæsth. Soc. J.*, *7*, 136 (April). 6 refs.

PHYSIOLOGY AND PHARMACOLOGY

Hyperventilation with oxygen – a possible cause of cerebral hypoxia. SUGIOKA, K. and DAVIS, D. A. (1960) *Anesthesiology*, *21*, 135 (March). 66 refs.

Effects of barbiturates on skeletal muscle function. BORGMAN, R. L., WALKER, T. R., PITTINGER, C. B. and LONG, J. P. (1960) *Anesthesiology*, *21*, 150 (March). 4 refs.

Endotracheal intubation: effects on blood pressure and pulse rate. WYCOFF, C. C. (1960) *Anesthesiology*, *21*, 153 (March). 4 refs.

A controlled clinical evaluation of two new analgesics, phenazocine and phenampromid. DEKORNFELD, T. J. and LASAGNA, L. (1960) *Anesthesiology*, *21*, 159 (March). 7 refs.

Cyclopropane arrhythmias in the cat: their cause, prevention and correction. ROBBINS, B. H. and THOMAS, J. D. (1960) *Anesthesiology*, *21*, 163 (March). 10 refs.

The electrocardiogram during anesthesia and surgery. CANNARD, T. H., DRIPPS, R. D., HELWIG, J. and ZINSSER, H. F. (1960) *Anesthesiology*, *21*, 194 (March). 13 refs.

Preliminary data on experimental electronarcosis induced with apparatus of the scientific research institute of experimental surgical apparatus and instruments. ANAN'EV, M. G., *et al* (1960) *Anesthesiology*, *21*, 215 (March). 13 refs.

Measurement of blood oxygen tension. BISHOP, J. M. (1960) *Proc. Roy. Soc. Med.*, *53*, 177 (March). 22 refs.

Measurement of blood carbon dioxide tension. NUNN, J. F. (1960) *Proc. Roy. Soc. Med.*, *53*, 180 (March). 12 refs.

Local analgesic drugs. BRYCE-SMITH, R. (1960) *Brit. Med. J.*, *1*, 1039 (April 2).

Monitoring of ventilation by integrated diaphragmatic electromyogram. FINK, B. R., HANKS, E. C., HOLADAY, D. A. and NGAI, S. H. (1960) *J. Amer. med. Ass.*, *172*, 1367 (March 26). Determination of carbon dioxide threshold in anesthetized man. 7 refs.

Comparison of toxicity of intravenously given local anesthetic agents in man. FOLDES, F. F., MOLLOY, R., MCNALL, P. G. and KONKAL, L. R. (1960) *J. Amer. med. Ass., 172*, 1493 (April 2). 20 refs.

Plasma levels of epinephrine and norepinephrine – anæsthetic significance. HAMELBERG, W., SPROUSE, J., MAHAFFEY, J. E. and RICHARDSON, J. A. (1960) *J. Amer. med. Ass., 172*, 1596 (April 9). 13 refs.

The respiratory response to carbon dioxide in health and disease. BRODOVSKY, D., MACDONELL, J. A. and CHERNIACK, R. M. (1960) *J. Clin. Invest., 39*, 724 (May). 11 refs.

Temperature changes in children during general anæsthesia. HARRISON, G. G., BULL, A. B. and SCHMIDT, H. J. (1960) *Brit. J. Anæsth., 32*, 60 (February). 7 refs.

Effect of Phenol on nervous conduction. NATHAN, P. W. and SEARS, T. A. (1960) *J. Physiol., 150*, 565 (March). 9 refs.

The cardiovascular effects of azeotropic halothane-ether. WYANT, G. M., MERRIMAN, J. E., HARLAND, J. H. and DONALDSON, H. V. (1960) *Canad. Anæsth., Soc. J., 7*, 91 (April). 6 refs.

Double blind study of phenothiazines used in pre-anæsthetic medication. DOBKIN, A. B. and PURKIN, N. (1960) *Canad. Anæsth. Soc. J., 7*, 158 (April). 16 refs. (Promethazine (phenergan) promazine (sparine) proclorpherazine (stemetil) and levopromazine were studied.)

Further studies of the influence of carbon dioxide on neuromuscular blocking agents in the cat. PAYNE, J. P. (1960) *Brit. J. Anæsth., 32*, 202 (May). 8 refs.

Mechanisms leading to lung œdema in pulmonary embolisation. KABINS, S. A., FRIDMAN, J., NEUSTADT, J., ESPINOSA, G. and KATZ, L. N. (1960) *Amer. J. Physiol., 198*, 543 (March). 13 refs.

Effects of d-tubocurarine on the submandibular salivary gland. ELWELL, L. H. (1960) *Amer. J. Physiol., 198*, 621 (March). 15 refs. (A local effect traceable to histamine release occurs.)

Role of sympathetic nervous system in CCl_4 hepato-toxicity. CALVERT, D. N. and BRODY, T. M. (1960) *Amer. J. Physiol., 198*, 669 (March). 33 refs. (Vasodilators reduce toxicity very markedly.)

Release of catechol amines from adrenal medulla by CCl_4. BRODY, T. M. and CALVERT, D. N. (1960) *Amer. J. Physiol., 198*, 682 (March). 14 refs.

Mode of action of local anæsthetics. WATSON, P. J. (1960) *J. Pharmacy & Pharmacol., 12*, 257 (May). 149 refs.

Anoxia and ventricular fibrillation: with a summary of evidence on the cause of fibrillation. BURN, T. H. and HUKOVIC, S. (1960) *Brit. J. Pharmacol., 15*, 67 (January). 22 refs.

RESUSCITATION

A critical assessment of the use of blood transfusions during major gastric operations. WILSON, B. J. and ADWAN, K. O. (1960) *Arch. Surg., 80*, 760 (May). 17 refs.

A consideration of indications for pre-operative transfusions based on analysis of blood volumes and circulating proteins in normal and malnourished patients with and without cancer. PEDEN, J. C., MAXWELL, M., OHIN, A. and MOYER, C. A. (1960) *Ann. Surg., 151*, 303 (March). 46 refs.

The effects of venoarterial pumping for circulation support. DICKSON, J. F., HAMER, N. A. J. and DOW, J. W. (1960) *Arch. Surg., 80*, 830 (May). 12 refs. Animal experiments.

Overtransfusion in the operating room. COSTELLO, C. J. (1960) *Arch. Surg.*, *80*, 843 (May). 2 refs.

Mouth to mouth resuscitation. TOMASHEFSKI, J. F. and OLIVER, T. K. (1960) *J. Amer. med. Ass.*, *172*, 1888 (April 23). 10 refs.

TREATMENT AND MEDICATION

Intermittent positive pressure respiration and therapeutic bronchial lavage in intractable status asthmaticus. BROOM, B. (1960) *Lancet*, *1*, 899 (April 23). 10 refs.

Administration of drugs before anæsthesia. MUSHIN, W. W. (1960) *Brit. med. J.*, *1*, 1558 (May 21).

Effect of dextro propoxyphene, meperidine and codeine on post-operative pain. VAN BERGEN, W. S., NORTH, W. C. and KARP, M. (1960) *J. Amer. med. Ass.*, *172*, 1372 (March 26). 12 refs.

Disturbance of body fluids – a symposium (1960) *Post. grad. med. J.*, *36*, 69–134 (February). Numerous references.

A method for assessing the efficacy of oral analgesics: Its applications and limitations. DUNDEE, J. W. (1960) *Brit. J. Anæsth.*, *32*, 48 (February). 9 refs.

Meprobamate in tetanus. ZUCK, D. (1960) *Brit. J. Anæsth.*, *32*, 186 (April). 16 refs.

EDUCATION & RESEARCH TRUST FUND

The Council of the Association will be pleased to consider applications from Fellows or Members needing financial assistance for educational or research purposes. Applicants for travel grants should state the objects of their proposed visits and submit fully detailed itineraries. Applicants for grants for research should state not only the objects of their investigations but also their proposed methods of experimentation. In all cases applicants should provide evidence that they have been unable to procure any or all of the necessary assistance from local sources. Any apparatus acquired through this fund remains the property of the Association of Anæsthetists and must be made available for the use of other Fellows or Members if subsequently required.

Obituries

Dr Cecil Hughes, consulting anæsthetist to King's College Hospital, died at his home in Hove, Sussex, on 2nd May. He was eighty-one years of age.

Cecil Hugh Myddleton Hughes was born in London in 1879, and was educated at Westminster School and at the Westminster Hospital, qualifying MRCS, LRCP in 1903, and graduating MB, BS two years later. After holding resident posts he was, in 1903, appointed administrator of anæsthetics at Westminster Hospital and he remained there until 1913 when he joined the honorary staff of King's College Hospital, which had just been removed to Denmark Hill, as assistant anæsthetist. On the retirement of J. F. W. Silk in 1921, Hughes became senior anæsthetist and lecturer in anæsthetics. When he resigned in 1936, several years before he was due to retire under the age limit, he was appointed consulting anæsthetist. He was also anæsthetist to the Seamen's Hospital, Greenwich, the Samaritan Free Hospital, the National Dental Hospital and the Bethlem Royal Hospital. During the 1939 war he returned to work at the Leatherhead Emergency Hospital in the King's sector.

Hughes was one of a small company of medical men who served in both the Army and the Royal Navy. During the South African war, while he was still a junior medical student, he joined the South African Field Force and was attached to the Imperial Yeomanry Hospital. Here he had the extraordinary experience of being captured by the famous General de Wet in person, and of managing to make his escape. At the end of the war he was awarded the South African medal with three clasps. In the war of 1914–8 he served as consultant anæsthetist to the Royal Navy with the temporary rank of Surgeon-Commander. He was posted to the Royal Naval Hospital, Chatham, where he worked with a famous King's surgeon, the late Sir William Watson Cheyne, whom Lister had brought with him as his house surgeon when he left Edinburgh for King's in 1877. At the end of the war, Hughes was appointed OBE.

Cecil Hughes was both secretary and president of the Anæsthetics Section of the Royal Society of Medicine and he was elected a Foundation Fellow of the Faculty of Anæsthetists in 1948. It is not generally realised that Hughes was primarily responsible for the foundation of the Hickman Medal. The initial suggestion that the centenary of Hickman's death should be suitably commemorated came from the Reverend F. Wayland Joyce but, when the interest of the Anæsthetics Section was aroused, it fell to the lot of Hughes, as secretary, to make suggestions to the committee as to the best way of commemorating the occasion. He was responsible for the detail of arrangements and it was a great joy to him that the memorial tablet in Bromfield Church was dedicated by another King's man, the late Sir St Clair Thomson, then the President of the Royal Society of Medicine.

It is difficult for one who is thirty years his junior to assess Hughes' worth as an anæsthetist. That he belonged to the old school of the 'rag and bottle' is undeniable, yet, at a time when deep ether anæsthesia was regarded as the safest and the best method for general surgery, he recognized the dangers and refused

to employ it. He was no scientist but an artist, a master of the art of light anæsthesia, almost of analgesia. A small wooden prop having been inserted between the patient's teeth, the Clover inhaler was gently approached to the face and was removed when the index pointer had reached the first quarter on the scale. Then a Schimmelbusch mask, covered with a single layer of lint, was substituted and there commenced the careful, slow dropping of minute amounts of chloroform and ether mixture. A gentle opening of the eye, a delicate touch upon the cornea, and the patient was adjudged to be ready for operation. Endotracheal tubes were only employed when absolutely essential and then it was always the old-fashioned grey gum elastic catheter. If abdominal relaxation was required, Hughes employed spinal analgesia. He took a great interest in his students and housemen and was a good teacher, being particularly insistent upon an exact knowledge of the signs of anæsthesia.

Hughes was a quiet man, very gentle in his manner, kindly and courteous to everyone. No one who ever experienced his hospitality could possibly forget his perfection of manners. He was the perfect host whose sole aim appeared to be to put his guest, often a junior and nervous houseman, entirely at his ease. This charming characteristic persisted throughout his long life and, only six months ago, the writer had the privilege of experiencing once again the easy, light conversation, the eager attention to his comfort, that he so well remembered from the days when he was Cecil Hughes' houseman, a quarter of a century ago.

Hughes married late in life. His marriage to Anne Athol, the widow of Dr W. Rivers Pollock, was an ideally happy one. His wife's death, in 1958, was a sad loss from which he never really recovered.

FFC

Dr A. E. W. Idris, formerly Senior Consultant Anæsthetist to the Middlesex and Hampstead General Hospitals, died after a brief illness on 21st May 1960, in his 71st year.

Arthur Ernest Williams Idris was born on 9th September 1889 and in 1908 he entered the Medical School at University College Hospital from which he qualified in 1913.

After holding house appointments at the old Great Northern Hospital and at Queen Mary's, Stratford, he joined the RAMC in 1915. A wound he received on service left him slightly lame.

After the war he became interested in anæsthesia and his aptitude for this speciality was recognised by his appointment in 1922 as Assistant Anæsthetist to the Middlesex Hospital, where he had previously been Resident Anæsthetist.

Appointments to many other hospitals soon followed; these included the National Dental, the Queen's at Sidcup, the Ministry of Pensions Hospital at Orpington, the Hostel of St Luke and the Hampstead General.

Meanwhile, he became Anæsthetist to the Dental Department at the Middlesex in 1927, before his promotion to the full Honorary Staff of that hospital in 1931.

The demands of private practice forced him to resign from many of these posts, but he was never too busy to attend St Luke's Hostel for the Clergy and he retained a deep affection for the Hampstead General, which he served for the rest of his professional life, becoming Senior Anæsthetist there after the second world war.

Although trained in the 'old school' of anæsthesia, he was receptive to new ideas and was an early practitioner of endotracheal and intravenous anæsthesia. He was also largely instrumental in introducing anæsthetic machines into routine use at the Middlesex.

Imperturbable in the operating theatre, he was much in demand for very major surgery in bad risk cases, while in routine work his dexterity at 'blind' nasal intubation and the rapid induction of inhalational anæsthesia was widely appreciated.

A member of the Anæsthetic section of the Royal Society of Medicine since 1924, he became a Founder Member of the Association of Anæsthetists in 1932, a Fellow in 1946 and a Senior Fellow in 1955. Granted the Diploma of Anæsthetics in 1935, he was elected FFA in 1948.

With the dispersal of the Hospital services in the 1939–45 war, he was posted to the EMS sector hospital at Tindal House in Aylesbury, but at the conclusion of hostilities he returned to the main hospital, where he became Senior Anæsthetist in 1946. In 1950 he was appointed Consultant Anæsthetist to the Ministry of Pensions.

In the early thirties he was involved in a fire and explosion in the operating theatre, the cause of which was never satisfactorily elucidated – as only low voltage electrical equipment was being used and the anæsthetic technique was accepted practice at that time.

While this unfortunate incident was of indirect benefit to the specialty in that it focused attention on the explosion hazard, it resulted in Idris himself being badly burnt and the explosion exacerbated his long-standing deafness.

The latter disability became severe towards the end of his career and in consequence he tended to avoid committees and professional meetings. Apart from this handicap he enjoyed robust health and was never known to wear an overcoat even in the depths of winter.

In 1954 he retired from his hospital appointments and from active practice devoting his attention to the garden of his home on the Thames near Abingdon and to his collection of antique furniture. His knowledge of this subject was great and he was, in fact, an expert carpenter and cabinet maker.

Ernest Idris will be mourned in particular by his juniors who will always be indebted to him – not onl for technical instruction and much good advice – but also for practical help, unbounded encouragement and loyal support during their resident appointments and in their subsequent careers.

Both they and the many generations of Middlesex students, to all of whom he was affectionately known as 'Pop' – to him they were always 'Laddie' – will gratefully remember his kindness, his warm humanity and unfailing good humour.

To his widow we extend our deep sympathy.

OPD

We very much regret to report the death of **Sir Gordon Gordon-Taylor** on 3rd September 1960 at the age of 82.

Many excellent obituary notices have already appeared of this doyen of British surgery and it would be out of place to add to them here. It should be recalled,

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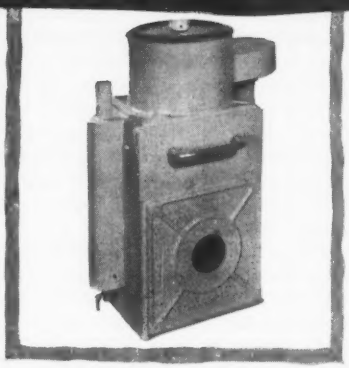

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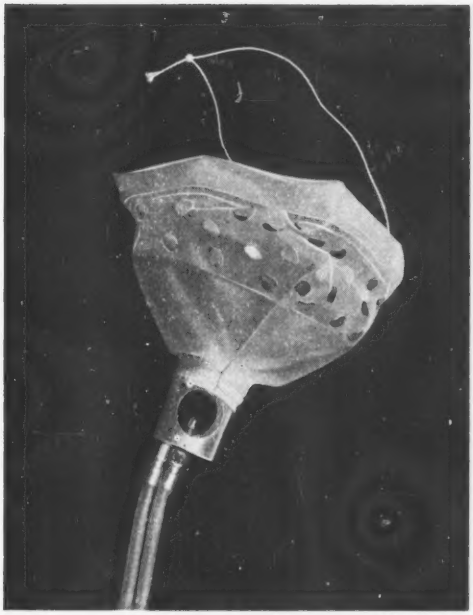

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*CAMPBELL, E. J. M. (2nd July 1960) *Lancet*, ii, 12.

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however, that 'G.T.' took a great interest in the advance of anæsthesia, an interest which was recognized by his election to Honorary Fellowship in the Faculty of Anæsthetists in 1957. He contributed an excellent account of the life of his late friend and colleague Dr Raymond Apperly in these columns in April of this year. Many of our London readers will miss the distinguished figure with a flower in his buttonhole and no overcoat, as he strode the streets on foot and who met his death while so doing.

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Book Reviews

HYPNOTISM FOR MEDICAL AND DENTAL PRACTITIONERS

A. A. MASON

Pp. 223. VIII Photographic Plates. Secker & Warburg, London. Price 30s.

Several books on medical hypnosis are now available and this is one of the best. Dr Mason is well qualified to be the author as he has devoted a considerable time to the subject and has also worked as an anæsthetist. Many readers will have seen some of his convincing demonstrations. He treats the whole subject as scientifically as is possible when so much is problematical and he avoids most of the usual jargon of the psychiatrist. Much space is give to detailed techniques of hypnotic induction and many applications to medicine, surgery and anæsthesia are suggested.

Dr Stanley D. Perchard has contributed a chapter on Hypnosis in Obstetrics and Mr K. Dawson Watts one on Hypnosis in Dental Surgery. There is a preface by the late Professor Alexander Kennedy of the Department of Psychological Medicine of the University of Edinburgh. The book is excellently printed and bound.

A PRACTICE OF GENERAL ANÆSTHESIA FOR NEUROSURGERY

ROBERT I. W. BALLANTINE and IAN JACKSON

Pp. viii+152. Figs 68. J. & A. Churchill Ltd, London. Price 27s. 6d.

Various monographs on particular aspects of anæsthesia have appeared in recent times and this one dealing with the problems of neurosurgery is most welcome.

The authors have found, as have so many before them, that simple techniques carried out with meticulous care and attention to detail, give quite as good results as 'advanced' and complicated methods with their attendant hazards. The reviewer well remembers seeing a demonstration by one of the authors of anæsthetizing two consecutive patients for removal of protruded intervertebral discs. The technique was the simple one of endotracheal nitrous oxide, oxygen, trichlorethylene with local infiltration of 1 in 500,000 adrenaline. The operative fields were so ischæmic that it was difficult to persuade the onlookers that some potent hypotensive drug had not been injected. Actually the systolic pressures never fell below 120mm/Hg. The authors' attitude to induced hypotension might, with advantage, be shared by all anæsthetists—'while the method must never be used for the convenience of the surgeon, if it facilitates his work to such an extent that it affects the success of the operation and increases the patient's chance of survival, then it is justifiable'. This sane and balanced outlook marks the whole of the text which covers the anæsthetic techniques for practically every neurosurgical operation and investigation in use at the present time. The precise

positioning of the patient for every procedure is rightly emphasised as he may have to remain in it for many hours. Readers will find much of special interest including the anæsthetic records of the eight hour operation for the separation of the famous craniopagus twins.

There is little doubt that the late Dr Z. Mennell would have welcomed this book as the logical development of his pioneering work. Mr J. E. A. O'Connell has written a foreword and the volume is dedicated to the memory of Dr Brian Rait-Smith, a former anæsthetist to the neurosurgical unit at St Bartholomew's Hospital who himself died from rupture of a cerebral aneurysm. The book is beautifully produced, the line drawings of Dr A. M. Hall-Smith being exceptionally clear and informative. There is a good index and a list of references rounds off each chapter.

NITROUS OXIDE IN DENTISTRY ITS DANGER AND ALTERNATIVES

J. G. BOURNE

Pp. x+181. Figs 23. Lloyd-Luke (Medical Books) Ltd, London. Price 30s.

This book is an elaboration of the author's well-known views that nitrous oxide and oxygen given in the dental chair is not as safe as is generally supposed and that the hypoxia which is inevitable with such a weak anæsthetic, sometimes accompanied by 'fainting', may cause permanent cerebral damage. Dr Bourne believes that the ideal anæsthetic for dentistry is cyclopropane and in order to eliminate the explosion hazard, it should be mixed with nitrogen and oxygen and given from a 6 litre bag. The main thesis is well set out and a very complete list of references to back it up is given at the end of the book.

Possibly the most important information is given in Appendix F. Five dental anæsthesia machines regularly serviced and in use were tested regarding actual performance. It was found that 'the wide discrepancies between the oxygen settings and the amounts of oxygen delivered, make the provision of a graduated oxygen scale unwarrantable'. For example, with the oxygen control set at 10 per cent, the oxygen actually delivered varied from 1.7 per cent to 20.1 per cent. This is a very disquieting observation and it seems remarkable that with so many years' experience, it has proved impossible for manufacturers to supply apparatus of reasonable accuracy. Here surely, lies the cause of some at least of the complications following nitrous oxide, oxygen anæsthesia in dentistry. At the present time it would seem wise to ignore the alleged mixture settings on such machines and rely entirely upon the signs shown by the patient.

This is a very interesting and thought-provoking book.

ANÆSTHETICS FOR MEDICAL STUDENTS

GORDON OSTLERE and ROGER BRYCE-SMITH

Foreword by C. LANGTON HEWER

Pp. viii+116. Fourth edition. J. & A. Churchill Ltd, London. Price 12s.

The popularity of this small book for undergraduates, is vouched for by the fact that four editions and two reprints have been called for in eleven years. The text remains substantially the same, but it has been brought up-to-date and a small section on induced hypotension has been added.

HYPOTHERMIA IN SURGICAL PRACTICE

K. E. COOPER and D. N. ROSS

Pp. xi+116. Figs 40. Cassell & Co. Ltd, London. Price 18s. 6d.

No-one will deny that the introduction of hypothermia has been one of the factors responsible for the recent great advances in cardiac and neurosurgery. So far as the reviewer is aware, this is the first book published by British workers which deals with all aspects of this important subject. The two chief authors are assisted by Dr B. A. Sellick, who deals with physiological considerations and the pharmacology of the drugs used, Mr L. S. Walsh (hypothermia in neurosurgery) and Dr V. Wynn (metabolic problems). The various methods used for the production of moderate, deep and selective hypothermia, with and without assisted circulation, are dealt with lucidly, and illustrated with clear diagrams and photographs. A useful chapter on unintentional hypothermia and its treatment is included. A good list of alphabetical references is given together with a short index. The authors have succeeded in producing a most readable introduction to a subject which is developing with great speed.

MEASUREMENT OF SUBJECTIVE RESPONSES

HENRY K. BEECHER

Pp. xvi+494. Oxford University Press. Price 102s.

This is a remarkable book, in which the author seeks to show that subjective sensations and symptoms, instead of being described vaguely, can be accurately measured and that the effects of drugs upon them can also be estimated quantitatively. The first section is devoted to an exhaustive discussion on the measurement of pain and other subjective responses. The rest of the book deals with the measurement of the effects not only of drugs, but of many other factors on these responses. For instance, methods are suggested to measure the extent of sedation, mental clouding and hypnosis, and the quantitative effects of narcotics on hunger, nausea, itching, anxiety and many other hitherto ill-defined conditions. Much of the work described is of interest to anæsthetists, especially that dealing with alterations in the pain threshold, drug interactions, potentiation and 'synergism'. The whole effort of the author is to convert much of the art of anæsthesia into a science, and thus to ensure more constant and predictable results than are at present possible.

The book is admirably documented, and we are glad to note that the 'Harvard system' of references is not used, but the much clearer one of small superior numerals which refer to the authorities' names in alphabetical order at the end. There are over one thousand of such references, and as thirty-three of these are under the author's name, it is clear that he has done a great deal of original work on this complex subject. In addition to the list of references, there is a comprehensive index.

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★ *Facilities for authors and lecturers.* We will gladly loan photographs, diagrams, printing blocks and actual equipment. In some cases sectioned models, film strips and lantern slides can also be made available.

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Association News

We have pleasure in announcing the appointment of the following to the **Panel of Advisers** to '*Anæsthesia*': Dr J. Clutton-Brock, Dr G. A. Rawlins, Dr R. D. Robertson.

The following **Commonwealth Correspondents** have also been appointed: Dr H. Grant-Whyte (Durban, S. Africa), Professor O. V. S. Kok (Pretoria, S. Africa), Dr N. R. James (Melbourne, Australia), Dr J. R. Ritchie (Dunedin, New Zealand) and Dr J. G. Robson (Montreal, Canada).

The **Scottish Society of Anæsthetists** held its Annual General Meeting at St Andrews from 6th–8th May 1960.

The Office-Bearers for 1960–1 are as follows:

PRESIDENT	Dr Andrew Tindal (Glasgow)
VICE-PRESIDENT	Dr J. W. L. Bain (Aberdeen)
HON. SECRETARY	Dr M. Shaw (Dept of Anæsthetics, Victoria Infirmary, Glasgow)

Dr Tindal gave as his Presidential Address 'Anæsthesia – things that may happen', in which he gave his own shaping of the future of our specialty. The Registrars' Prize was won by Dr G. R. Dow, Western Infirmary, Glasgow, with his paper on 'The indications for steroids in anæsthesia'. The Guest Speaker was Professor Sir Dugald Baird, Aberdeen; his address on Obstetric Anæsthesia and Analgesia was a survey of the drugs and techniques employed during his own professional career. Dr Deidre Gillies, on a short visit from Montreal, was welcomed and invited to address the meeting.

The **Liverpool Society of Anæsthetists** has arranged the following programme for the session 1960–1.

1960

Friday 7th October 8 pm. Ordinary General Meeting at Liverpool Medical Institution. *Speaker:* Dr H. J. V. Morton. Clinical experiments galore.

Friday 11th November 8 pm. Ordinary General Meeting at Liverpool Medical Institution. Brains trust on pediatric anæsthesia. *Chairman:* Dr J. B. Hargreaves. *Panel:* Professor Gray, Dr R. W. Cope, Dr Robert M. Smith, Dr G. J. Rees.

1961

Friday 20th January 8 pm. Ordinary General Meeting at Liverpool Medical Institution. *Speaker:* Dr A. H. Galley. Dental anæsthesia, past, present and future.

Thursday 16th February 8 pm. Joint Meeting with Liverpool Medical Institution at Liverpool Medical Institution. *Subject:* The role of the anæsthetist in the management of intractable pain. *Speakers:* Dr J. E. Riding, Therapeutic nerve blocks; Dr J. W. Dundee, Recent progress in drug therapy.

Friday 17th March 8 pm. Ordinary General Meeting at Liverpool Medical Institution. Papers to be presented by Registrars.

Thursday 20th April 8 pm. Combined Meeting with Anæsthetic Section of Manchester Medical Society at Liverpool Medical Institution. *Speakers:* Dr M. Swerdlow, Epidural studies; Dr A. L. Stead, Dental anæsthesia for children.

Friday 19th May 8 pm. Twenty-ninth Annual General Meeting at Liverpool Medical Institution.

Faculty News

Faculty of Anæsthetists, RCSI: Dr Geoffrey Organe, the Dean, represented the Faculty at the inaugural meeting of the Irish Faculty in Dublin on 8th July, and presented a congratulatory address and a copy of the recently published History of the Royal College of Surgeons by Sir Zachary Cope. One Honorary Fellow of the Faculty, Sir Ivan Magill, KCVO and two Fellows, the Dean and Dr John Gillies, CVO, have been elected to the Honorary Fellowship of the new Faculty.

Examinerships: Dr R. P. W. Shackleton is unable to take up the examinership for the Final FFARCS, announced in July and Dr G. Jackson Rees has been appointed to take his place. Professor W. W. Mushin has been appointed to examine in pharmacology at the Primary FFARCS examination to be held in Colombo in January.

Course for Consultants and S.H.M.O's.: The dates of this three-day course, which was mentioned in Faculty News in July, have been arranged from 24th–26th November 1960.

At a meeting held in the **Royal College of Surgeons in Ireland** on Friday 8th July 1960, the President of the College, Mr T. G. Wilson, inaugurated a new Faculty of Anæsthetists. The first Board of the Faculty consists of:

DEAN	Dr Thomas James Gilmartin
VICE-DEAN	Dr Victor Ormsby McCormick
HON. SECRETARY	Dr Joseph Augustine Woodcock
MEMBERS	Dr Patrick Joseph Drury Byrne
	Dr Patrick Joseph Nagle
	Dr Ethel Sheila Kenny
	Dr Paul Finbar Murray
	Dr Geoffrey Raymond Davys
	Dr John Wharry Dundee
	Dr Samuel Harold Swan Love

After admitting the Board members as Foundation Fellows in the Faculty, the President also admitted the following Foundation Fellows: Drs Sachindra Nath Basu, Kathleen Bayne, William Bingham, John Boyd, George John Cecil Brittain, Daniel Gerard Coleman, Walter Samuel Davis, Kevin Bernard Glynn, Thomas Francis Heavey, John Cecil Hewitt, Margaret Marion Vida Lemon, John Alexander MacAulay, Mary Josephine Ahern O'Connor, Margaret Silver Deane Oliver, Lady Frances Edna Read, Richard William Shaw and Maurice Alexander Woods.

The following Foundation Fellows were also conferred *in absentia*: Drs James Daniel Bourke, Arthur Barclay Bull, Francis MacDermot Byrn, Anthony Halton Kasasian, Ordino Victor Steyn Kok, Zoltan Lett, Brendan Vincent Lyne, Paul Ronald Mesham, Maureen Murphy and David Lindsay Scott.

The Honorary Fellowship in the Faculty was then conferred by the President on Dr John Gillies, who was introduced by the Vice-Dean, Dr McCormick and upon Dr Geoffrey Stephen William Organe, who was introduced by Dr Drury Byrne.

Commonwealth and Foreign News

The **American Society of Anesthesiologists** has moved its headquarters to 515 Busse Highway, Park Ridge, Illinois, U.S.A.

We congratulate **Dr C. R. Ritsema van Eck** on being appointed Professor of Anæsthesia in the University of Groningen, Holland.

At a recent meeting of the **Brazilian Association of Anæsthetists** Dr J. P. Payne lectured on halothane and gave demonstrations on its use.

First International Pharmacological Meeting, Stockholm, Sweden, 22nd–25th August 1961.

Held under the auspices of the Section of Pharmacology of The International Union of Physiological Sciences. The programme is designed to promote attendance by members of the International Union of Biochemistry visiting the Fifth International Congress of Biochemistry.

Scientific programme. It has been decided to restrict the scientific programme to topics under the general heading of 'Mode of action of drugs'. Depending upon the availability of speakers, main topics will be chosen from the following titles: 'Pharmacological control of release of hormones, including antidiabetic drugs': 'Effects of drugs on synthesis and mobilization of lipids': 'New aspects of cardiac pharmacology': 'Drugs and membranes': 'Methods for the study of pharmacological effects at the cellular and subcellular levels': 'Metabolic factors controlling duration of drug action': 'Pharmacological analysis of central nervous action'.

The convention will invite prominent workers in the respective fields to deliver introductory lectures. In addition to these, brief communications reporting original research will be accepted in so far as facilities permit and will be scheduled after the introductory lectures.

All enquiries to Secretariat of the First International Pharmacological Meeting, Secretary General, Dr Arvid Wretlind, Karolinska Institutet, Stockholm 60, Sweden.

The **Anæsthetic Section of the Belgian Society of Surgery** celebrates this year the tenth anniversary of its foundation. A scientific meeting will be held on 26th–27th November in the 'Benelux Room' of the Palais des Congrès in Brussels. The general subject will be Respiratory Resuscitation and particulars can be obtained from the Secretary of the Section, Dr A. Goldblat at the above address.

There are five vacancies for consultant anæsthetists at hospitals in **Durban, Natal.** Applicants should hold the FFARCS diploma. Particulars appear in an advertisement in the *Journal of the Medical Association of South Africa*, which can be seen at South Africa House, Trafalgar Square, London, WC2.

The second **World Congress of Anæsthesiologists** has just finished its meetings which were held at the Royal York Hotel, Toronto, under the presidency of Dr Harold Griffith of Montreal. About 1,700 delegates and associate delegates attended, representing twenty-three countries, and over one hundred and fifty papers were read, the majority in English, but some in French, German and Spanish. A simultaneous translation service was organised, each delegate being provided with a pair of head-phones and a small transistorised short-wave radio receiver, which received the translation into the language of his choice. During the week, panel discussions were held on Assisted and Controlled Respiration, Anæsthesia in Cardiac Surgery and on Teaching of Anæsthesia. In addition, twenty films on anæsthetic topics were shown at frequent intervals. The temperature during the Congress was up in the nineties and humidity was high, but this did not seriously detract from the success of the meeting.

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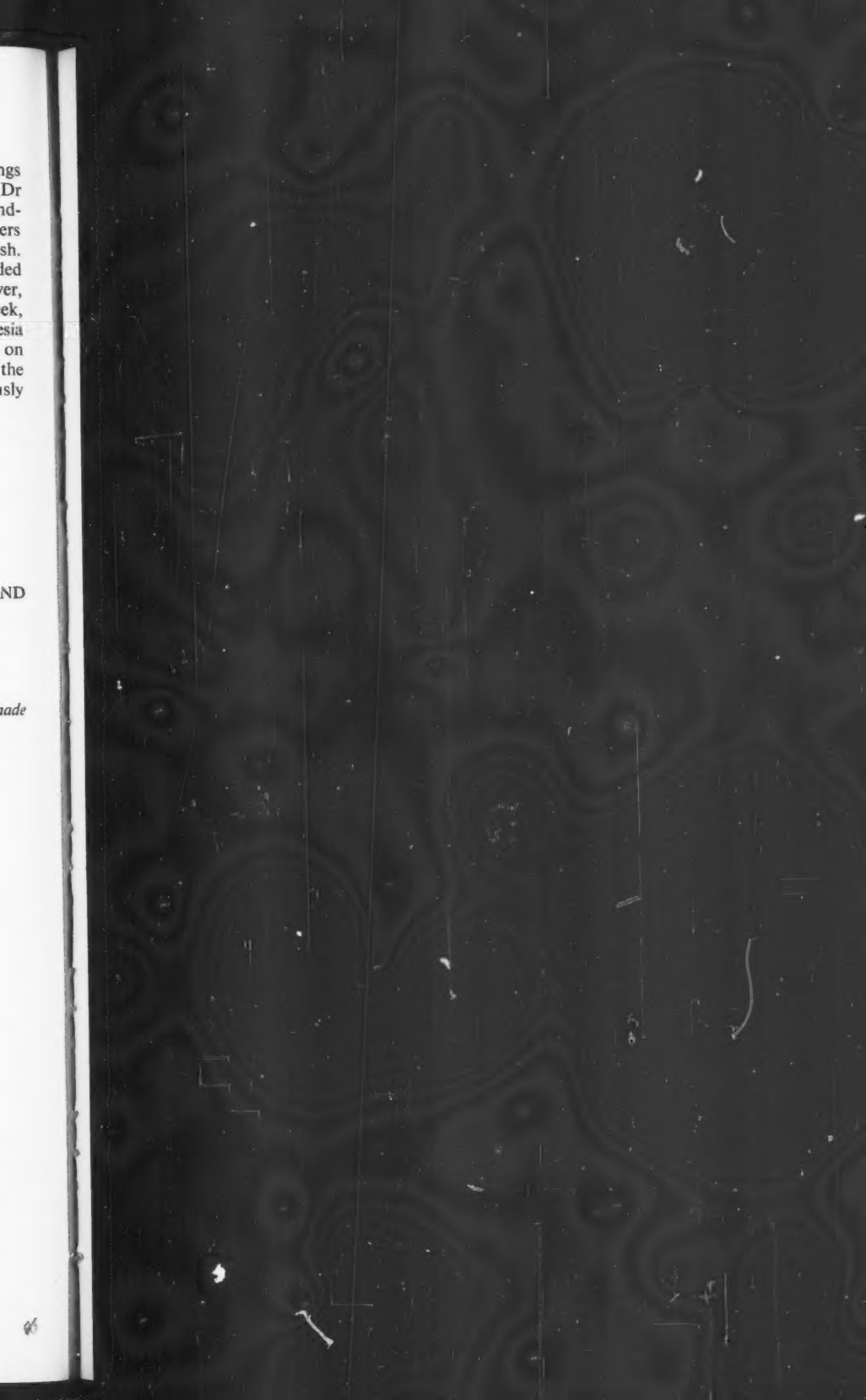
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